HYPEREMESIS GRAVIDARUM
AND
PREGNANCY OUTCOME

Dissertation submitted to

THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY

In partial fulfillment of the degree of

M.S. OBSTETRICS & GYNAECOLOGY

PSG INSTITUTE OF MEDICAL SCIENCES AND RESEARCH, COIMBATORE.

DEPARTMENT OF OBSTETRICS & GYNAECOLOGY

APRIL 2016

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CERTIFICATE

This is to certify that **Dr. S. SUJITHRA** REG NO : 221216454 postgraduate student (2012-2016) in the department of Obstetrics & Gynaecology, PSG INSTITUTE OF MEDICAL SCIENCES AND RESEARCH, Coimbatore has done this dissertation titled **"Hyperemesis Gravidarum & Pregnancy Outcome"** under the direct guidance and supervision of guide **Prof. Dr.T.V. CHITRA MD,DGO,DNB** in partial fulfillment of the regulations laid down by the **Tamilnadu Dr.M.G.R. Medical university**, Chennai, for the award of M.S., Degree in Obstetrics & Gynaecology.

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DECLARATION

I, **Dr. S.SUJITHRA**, solemnly declare that this dissertation **" HYPEREMESIS GRAVIDARUM AND PREGNANCY OUTCOME"** is a bonafide record of work done by me in the **Department of OBSTETRICS AND GYNAECOLOGY**, PSG Institute of Medical Sciences and Research,Coimbatore, under the guidance of **Prof. DR.CHITRA .T.V. MD,DGO,DNB.** This dissertation is submitted to "The Tamilnadu Dr.M.G.R.Medical University, Chennai " in partial fulfillment of the University regulations for the award of **MS Degree (Obstetrics & Gynaecology)**, Examination to be held in April 2016.

Place: Coimbatore

Date:

Dr.S.SUJITHRA

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Last but not the least I express my gratitude to all the patients for their cooperation for being a part of my study.

DR. SUJITHRA.S

PSG Institute of Medical Sciences & Research
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September 12, 2014

To
Dr S Sujithra
Postgraduate
Department of Obstetrics & Gynaecology
PSG IMS & R
Coimbatore

The Institutional Human Ethics Committee, PSG IMS & R, Coimbatore -4, has reviewed your proposal on 12th September, 2014 in its expedited review meeting held at IHEC Secretariat, PSG IMS&R, between 10.00 am and 11.00 am, and discussed your study proposal entitled:

"Hyperemesis gravidarum and pregnancy outcome"

The following documents were received for review:

1. Duly filled application form
2. Proposal
3. Informed consent forms
4. Data collection tool
5. CV
6. Budget

After due consideration, the Committee has decided to approve the study.

The members who attended the meetings at which your study proposal was discussed are as follows:

Name	Qualification	Responsibility in IHEC	Gender	Affiliation to the Institution Yes/No	Present at the meeting Yes/No
Dr P Sathyan	DO, DNB	Clinician, Chairperson	Male	No	Yes
Dr S Bhuvaneshwari	M.D	Clinical Pharmacologist Member - Secretary	Female	Yes	Yes
Dr Y S Sivan	Ph D	Member – Social Scientist	Male	Yes	Yes
Dr D Vijaya	Ph D	Member – Basic Scientist	Female	Yes	Yes

The approval is valid for one year.

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We request you to intimate the date of initiation of the study to IHEC, PSG IMS&R and also, after completion of the project, please submit completion report to IHEC.

This Ethics Committee is organized and operates according to Good Clinical Practice and Schedule Y requirements.

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Kindly note this approval is subject to ratification in the forthcoming full board review meeting of the IHEC.

Yours truly,

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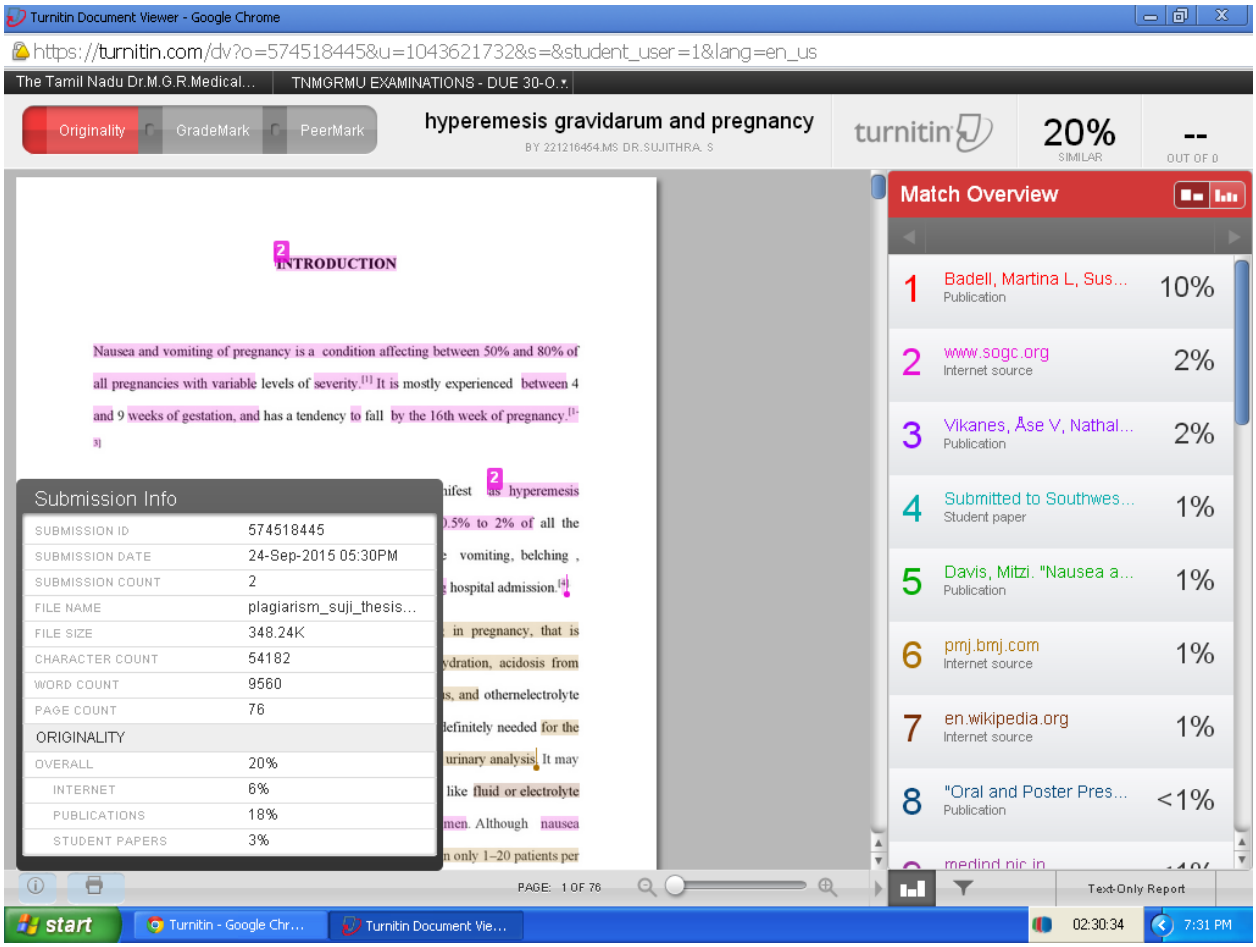
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INTRODUCTION
Nausea and vomiting of pregnancy is a condition affecting between 50% and 80% of all pregnancies with variable levels of severity.[1] It is mostly experienced between 4 and 9 weeks of gestation, and has a tendency to fall by the 16th week of pregnancy.[1-3]
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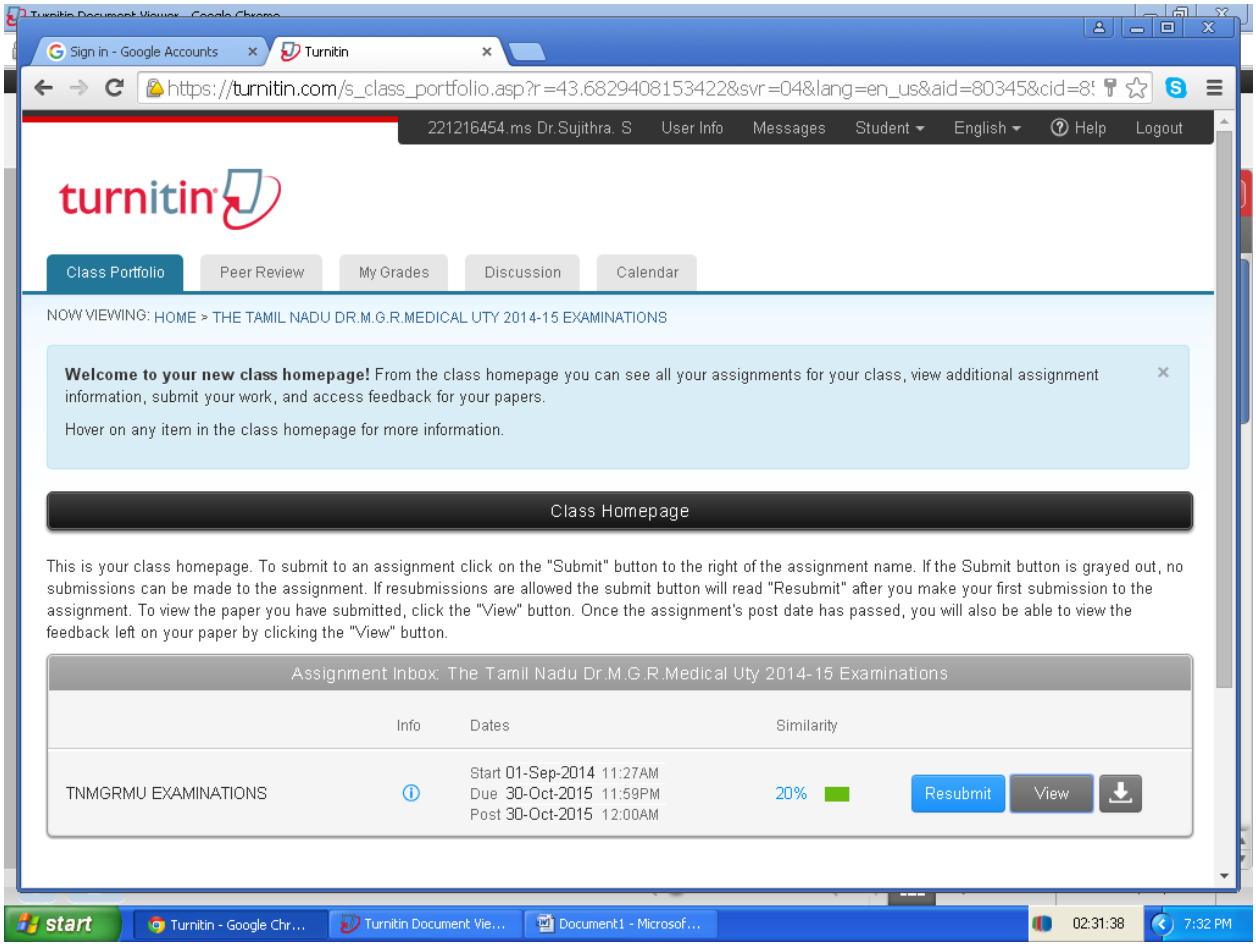
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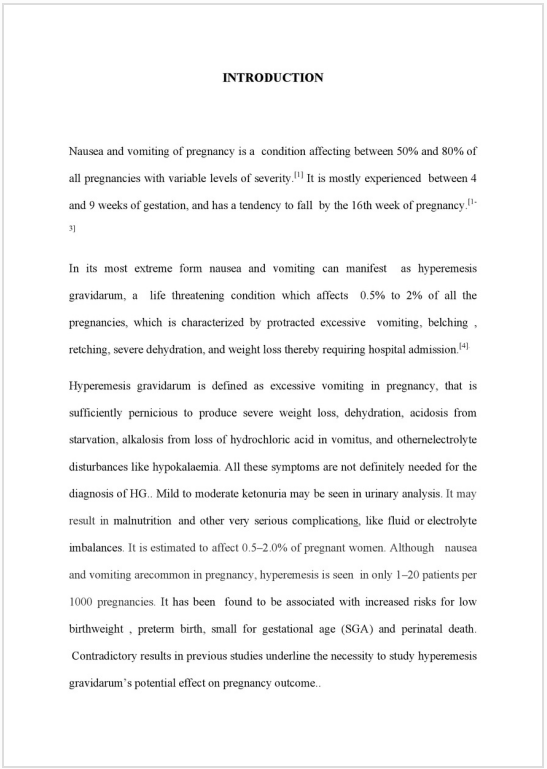

INTRODUCTION

Nausea and vomiting of pregnancy is a condition affecting between 50% and 80% of all pregnancies with variable levels of severity.[1] It is mostly experienced between 4 and 9 weeks of gestation, and has a tendency to fall by the 16th week of pregnancy.[1-3]

In its most extreme form nausea and vomiting can manifest as hyperemesis gravidarum, a life threatening condition which affects 0.5% to 2% of all the pregnancies, which is characterized by protracted excessive vomiting, belching , retching, severe dehydration, and weight loss thereby requiring hospital admission.[4]

Hyperemesis gravidarum is defined as excessive vomiting in pregnancy, that is sufficiently pernicious to produce severe weight loss, dehydration, acidosis from starvation, alkalosis from loss of hydrochloric acid in vomitus, and otherelectrolyte disturbances like hypokalaemia. All these symptoms are not definitely needed for the diagnosis of HG.. Mild to moderate ketonuria may be seen in urinary analysis. It may result in malnutrition and other very serious complications, like fluid or electrolyte imbalances. It is estimated to affect 0.5–2.0% of pregnant women. Although nausea and vomiting arecommon in pregnancy, hyperemesis is seen in only 1–20 patients per 1000 pregnancies. It has been found to be associated with increased risks for low birthweight , preterm birth, small for gestational age (SGA) and perinatal death. Contradictory results in previous studies underline the necessity to study hyperemesis gravidarum's potential effect on pregnancy outcome..

CONTENTS

INTRODUCTION

Nausea and vomiting of pregnancy is a condition affecting between 50% and 80% of all pregnancies with variable levels of severity.[1] It is mostly experienced between 4 and 9 weeks of gestation, and has a tendency to fall by the 16th week of pregnancy.[1-3]

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AIMS & OBJECTIVES

PRIMARY OBJECTIVE:

i. To study the maternal and fetal outcomes in pregnancies complicated by hyperemesis gravidarum (HG) as compared to controls.

SECONDARY OBJECTIVE:

i. To analyse the correlation between number of episodes of vomiting and maternal weight gain with the perinatal outcome.

ii. To study the association between dehydration and ketonuria with the maternal and fetal outcome.

iii. To analyse the association between duration of persistence of hyperemesis gravidarum in terms of gestational age with the maternal and fetal outcome.

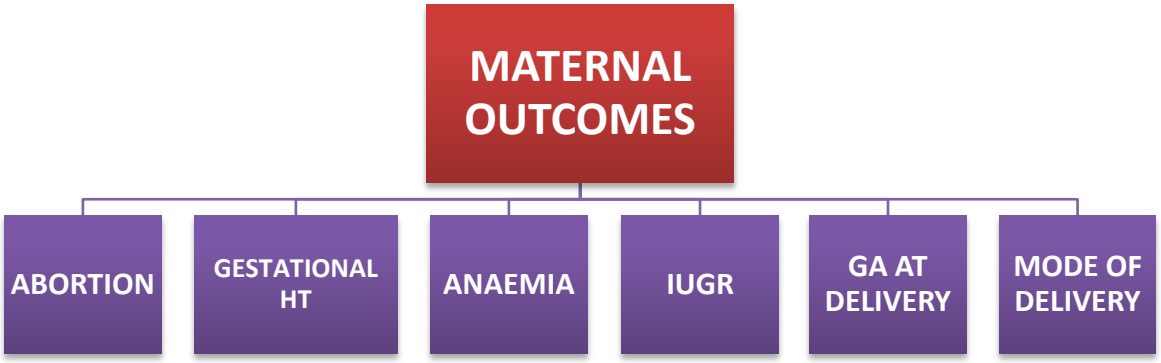
METHODOLOGY

This study is a prospective hospital based observational study conducted to analyse the impact of hyperemesis gravidarum on the maternal and fetal outcome. The study was conducted between June 2014 to July 2015 at PSG Institute of Medical Sciences And Research, Coimbatore.

All patients with singleton pregnancy diagnosed as hyperemesis gravidarum and with history of hyperemesis gravidarum in the present pregnancy who have been hospitalized and treated for the same were included in the study. Informed consent was obtained from each patient.

All these patients with hyperemesis gravidarum were admitted, examined for signs of dehydration, urine ketones were done, other causes of vomiting like acute gastroenteritis, intestinal obstruction etc., were ruled out. These patients were treated with intravenous fluids and antiemetics and were discharged on the 3rd or 4th day of hospital stay after tolerating orally and symptomatic improvement. These patients were followed up throughout pregnancy and the maternal and fetal outcome were evaluated and compared with the control group. Patients without hyperemesis

gravidarum were taken as controls and their maternal and fetal outcome were evaluated and the results were compared. In order to avoid bias in selecting the control the very next delivery following the delivery of a patient whose pregnancy was complicated by hyperemesis gravidarum were taken as control group. For each patient the following details were collected: age, obstetric score, gestational age at HG admission, no. of episodes, hydration status, presence of ketonuria, antenatal complications if any, gestational age at delivery, mode of delivery, birth weight, APGAR at 1st and 5th minute. The severity of hyperemesis gravidarum was assessed by the number of episodes of vomiting, gestational age at which the patient was hospitalised with HG and the presence of dehydration and ketonuria. The maternal outcomes evaluated were weight gain during pregnancy, abortion, gestational hypertension, gestational diabetes mellitus, anaemia, oligohydramnios, intrauterine growth retardation, gestational age of delivery and mode of delivery.



Neonatal outcomes evaluated were 1 and 5-minute APGAR score, birth weight and perinatal death.

STATISTICS:

The data was collected and entered in the SPSS data sheet. The data was analysed using SPSS 20 for descriptive statistics. The test variables were compared using Chi-square test for qualitative variables and Student's test for quantitative variables. The variables for which the association was statistically significant ($p < 0.1$) were introduced in a logistic model.

METHODOLOGY

Aim	• To study the maternal and fetal outcomes in pregnancies complicated by hyperemesis gravidarum (HG) as compared to controls. • To analyse the correlation between number of episodes of vomiting and maternal weight gain with the perinatal outcome. • To study the association between dehydration and ketonuria with the maternal and fetal outcome. • To analyse the association between duration of persistence of hyperemesis gravidarum in terms of gestational age with the maternal and fetal outcome.
Study design:	A Prospective Hospital based Observational study
Study Population:	All pregnant women in whom pregnancy was complicated by hyperemesis between june 2014-2015 were taken as cases and were followed up until delivery. Pregnant women with singleton pregnancy without hyperemesis in the same time period were taken as controls.
Sample Size:	Cases- 80 , control – 80
Inclusion Criteria	All pregnant women with singleton pregnancy with hyperemesis during the present pregnancy.
Exclusion Criteria:	• Multiple pregnancy • Molar pregnancy • Other medical or surgical causes of vomiting
Duration of the study	12 months
Study Period	June 2014 - July 2015

REVIEW OF LITERATURE

Epidemiology of nausea and vomiting in pregnancy (NVP):

Nausea and vomiting in pregnancy also known as morning sickness, affects about 75-80% of pregnant women [1]. Even before 4000 years, documentation of NVP is seen in the literature [2]. Symptoms usually start at around 5 weeks of gestation and usually ceases at 12weeks of gestation .But in 15% of pregnant women the symptoms may persist till delivery. Among the pregnant women, 50 % of them will have both nausea and vomiting and 25 % experience just nausea alone. Vomiting alone occurring as a symptom without nausea is very rare. Although " morning sickness " is the common term used to represent NVP, only around 17 % pregnant women have symptoms in the morning and the remaining experience symptoms the whole day[3].

Psychosocial support:

Although NVP is so common, it is a self limiting condition in most of the pregnant women during initial part of pregnancy without any long term negative impact on both mother and the fetus's health. But still it has a significant impact on personal and professional life of the mother during the early gestational period. It causes significant distress in most of the women. The emotional and physical stress due to this condition may inculcate a feeling of concern and anxiety in the mother about how the fetus might be affected. Most pregnant women are not comfortable with drug therapy and are much worried about the teratogenicity of the drugs used in the treatment of this condition.About 50 % of the pregnant mother who have nausea

and vomiting feel that, it might negatively affect their relationship and intimacy with their spouse and 55% of them are depressed[4].

There are rare case reports in the literature, where women had chosen abortion due to intolerance to symptoms of nausea and vomiting early in pregnancy [5]. Reports says, almost 50 % of working women believe that efficiency in their work is decreased due to nausea and vomiting and around twenty five percent of working women needed time off from their work as a result of these symptoms [6]. Severe nausea and vomiting in pregnancy is approximately estimated to cost about 130 million dollar per year, which has significant impact on health care system[7]. This high estimate is basically based mainly on the cost of hospitalisations and it does not include the financial cost to patients for their drug therapy, loss of productivity in job, doctors appointments, or impact on the quality of life.

Etiology and Pathophysiology of nausea and vomiting in pregnancy:

The exact etiology and pathophysiology of nausea and vomiting in pregnancy is still not clear and it's a matter of debate. Multiple risk factors had been proposed to be the cause of this condition. A number of theories had been proposed but none has been proved. The well renowned theories are the psychological and emotional predisposition theory, theory of evolutionary adaptation , theory of hormonal stimuli, and H. pylori infection theory. As nausea and vomiting has a range of spectrum of symptoms, assortment of factors play different roles at various degrees in each of the pregnant woman.

Theory of Psychological Predisposition:

In this hypothesis NVP was said to be a psychosomatic illness or a somatoform disorder, wherein women who had pregnancy related nausea and vomiting were not able to cope up with various stresses of pregnancy and which later converted into various physical symptoms[28]. Psychoanalytic theorists have explained pregnancy as time when pregnant mother is more prone to conversion disorders and any history of trauma might manifest as physical symptoms. The above said theory of NVP as psychiatric disorder was found to be difficult to confirm because previous studies were confounded by biases and mostly were not blinded or lacked in number of control subjects. Few reports in literature where the authors have concluded that the pregnant women had nausea and vomiting due to disharmony in relationships with their spouses or mothers or due to decreased feminity. In recent case control study, where in, women with hyperemesis gravidarum were compared with women without hyperemesis and with normal pregnancies[29] . using a scale for psychological conversion disorder, the authors found that there was no significant differences between women with hyperemesis gravidarum and the matched controls.

Few reports have identified association between NVP and psychological disorders such as anxiety, depression, and hysteria[16,30]. Furthermore personality disorders such as dependency and immaturity have been proposed to lead to nausea and vomiting as a maladaptive technique of coping during pregnancy. Secondarily it has also been used as a mode to obtain sympathy and attention among friends and family members[31].

It was also observed that women with NVP had disappearance of symptoms when they separate from family and the symptoms relapse when they return back to family environment, which proves the impact of psychological factors as a proposed etiology of NVP [32]. Thus factors like psychosocial and psychological factors can play a very important role in determining whether women experience only mild nausea or NVP that may progress to very severe symptoms or even to hyperemesis gravidarum[31].

Though evidences in literature are very less, the psychological and psychosocial influence of NVP has its own merit. For example patients with cancer who have received more than one chemotherapy cycles may develop nausea or vomiting after just passing the area where they received the treatment, scheduling appointments, or on going to their doctor's office. This shows that although nausea and vomiting in pregnancy has its own biologic cause related to pregnancy, psychological factors also play a very important role. Thus its unwise to really exclude role of psychological factors in NVP, rather more evaluation is required to identify the intricate relationship between biologic and psychological factors.

Theory of Evolutionary Adaptation:

As per this theory NVP in a mild form usually does not harm the mother nor the embryo and therefore it is considered as beneficial adaptation rather than disorder or disease . This theory explains the reason why some women develop short term dislike for certain foods alone in pregnancy. It also explains why nausea and vomiting in pregnancy is different among different cultures depending on the food

they consume. The authors in one of the study proposed that "nausea and vomiting of pregnancy is an intricate mechanism that probably evolved to serve a useful function: protecting the pregnant woman and embryo from food-borne infections and toxins .[33]

In another study the author has proposed that the pregnant women tend to avoid food borne teratogenic substances and abortifacients as a result of nausea and vomiting[34]. For example, the pregnant women have nausea and vomiting symptoms which rises during the first trimester, when the fetus is very susceptible to teratogenic effects of foreign substances. Moreover food which are avoided are the most potentially harmful toxic foods like the fish , poultry , eggs and meat which are more likely to contain bacteria and fungi [35]. Evidence also indicates that, women with nausea and vomiting have positive pregnancy outcomes, thus providing an additional support for the evolutionary adaptation[8-10]. However, the physicians should be very careful while applying this theory in patient management. With the belief that NVP is natural and beneficial should not lead to under management of symptoms. Whether this condition may be an adaptive mechanism for the mother and fetus , hyperemesis can be very distressful for the women both professionally and personally.

Theory of hormonal stimulus:

Another new theory suggests that changing levels of hormone mainly estradiol ,progesterone and Beta-HCG, may result in NVP, but mechanism remains unknown[36]. Gestational age at which the symptoms of nausea and vomiting peak is an very important factor to support the theory because, the onset of symptoms and its peak closely mimick the peak in the levels of the above said hormones.

Beta HCG :

There are two other observations in support of the involvement of beta-HCG in causation of nausea and vomiting. One is the association between the rise in the beta-HCG values and peak in the symptoms of nausea and vomiting. The second one is severe symptoms of nausea and vomiting seen in women with multiple gestations or molar pregnancy which involves elevated beta-HCG levels. In a review of 17 studies, 13 studies showed a relationship between beta-HCG and nausea and vomiting in pregnancy[37]. The failure in some of the studies could be due to varying biological activity of different isoforms of beta-HCG .Few isoforms of beta-HCG mimick thyroid stimulating hormone and are said to have more thyrotrophic activity[38]. Eight out of ten studies demonstrated a relation between transient hyperthyroidism and nausea and vomiting in pregnancy. Beta HCG is the stimulator of thyroid in pregnancy and therefore it is beta-HCG and not TSH that is responsible for the association between transient biochemical hyperthyroidism in pregnancy[39]. The accurate role of beta-HCG in causation of nausea and vomiting stays unclear. Increased levels of b-HCG alone do not predict the occurrence nor the severity of hyperemesis.

Estradiol:

Estrogen , mainly estradiol have been found to influence nausea and vomiting in pregnancy. Some authors showed that many women with hyperemesis had significantly raised levels of estradiol in first trimester when compared to the controls[14]. Decreased estradiol levels are seen with smoking cigarette; therefore

smoking women are less likely to develop hyperemesis gravidarum[40]. Exogenous estrogen also induce hyperemesis when given to non pregnant individuals. Women on oral contraceptive pills develop nausea and vomiting and are at high risk of developing hyperemesis in pregnancy[3]. Increased levels of estradiol during pregnancy although play a role in causing nausea and vomiting, it is not purely responsible ,nor predictive of the severity of symptoms of nausea and vomiting or in the development of hyperemesis.

Progesterone:

Changes in the hormonal levels in pregnancy disrupt the neuromuscular function of gastrointestinal tract thereby resulting in nausea and vomiting. Progesterone reduces the contractility of smooth muscles and causes dysrhythmias or delayed gastric emptying[14]. In a study, electrogastrograms of 32 women who were pregnant with hyperemesis showed that 26 women had dysrythmias like tachygastrias or bradygastrias[41]. The finding of above study was confirmed by a placebo trial in which the non- pregnant women were given oestrogen and progesterone in the levels equivalent to the first trimester of prenancy[42]. These non-pregnant women had slow wave dysrhythmias which was seen in pregnant women with hyperemesis..

Both the studies are in support of the hypothesis that oestrogens and progesterone at elevated levels in pregnancy may disrupt the normal gastric rhythm and might contribute to the causation of nausea and vomiting in pregnancy.

Helicobacter –Pylori Infection :

Infection with H.pylori has been found to be associated with NVP and hyperemesis gravidarum. A study in 1998 compared 105 women with hyperemesis gravidarum with 129 controls who were asymptomatic[43]. The results showed double the increase in antibodies to helicobacter pylori in those women with hyperemesis gravidarum. Another study in 2002 found that though most of women with hyperemesis were found to be positive for H.pylori antibodies, there was no correlation between serology positive and duration of the symptoms.

In one of the case report of 2 groups of women with hyperemesis gravidarum who were treated with erythromycin, both patients showed a marked improvement in symptoms after treatment[45]. Though erythromycin is mainly administered to treat Helicobacterpylori infection it also has prokinetic effect, which could improve nausea and vomiting caused by delayed emptying of gastric contents[46].

The diversity of the proposed hypothesis and lack of a definitive evidence makes the pathophysiology of NVP a complex phenomenon. Since the symptoms of women who is pregnant range from no symptoms to very frequent episodes and to hyperemesis gravidarum which requires hospitalization, a single causative factor is not likely. Hormonal changes, evolutionary adaptation, psychological changes and possible Helicobacter pylori infection can contribute to some extent to the development of nausea and vomiting in pregnancy.

Pregnancy outcome:

Few studies have reported that NVP is associated with favourable pregnancy outcome[8-10]. An interesting study conducted by group of investigators in 411 pregnant women about the pattern of hyperemesis its pregnancy outcome showed that women without nausea and vomiting were found to have a greater proportion of non viable fetus and low birth weight infants than those who had vomiting [8].

HG is said to be in correlation with low birthweight (LBW), premature birth (PTB), small-forgestational-age, prolonged hospitalisation for the new born and perinatal death [126-129]. A current meta-analysis on hyperemesis gravidaruam and pregnancy outcome , comprising of thirteen case control studies, 10 studies and one cross-sectional study, found that hyperemesis gravidarum was associated with 30% increased risk of preterm birth and small for gestational age, and 40% increased risk for low birth weight [127]. A historical Dutch cohort study among 1.2 million singleton births found that hyperemesis in pregnancy increases the risk of preterm birth by 18%, but did not find any association between hyperemesis gravidarum and small for gestational age and low bith weight [128]. The study also reports a mild difference in birthweight among babies born to women with HG and those without hyperemesis gravidarum[128]. In contrast, a Canadian cohort study of 156,000 singleton pregnancies found that women with Hyperemesis who gain weight less than 7 kg throughout pregnancy had a three times increased risk of preterm birth, almost three times increased risk fo low birth weight and a five times increased risk for 5 minute Apgar score less than 7 [126]. In women with HG and Weight gain more than or equal to 7 kg, there wasn't any increased risk of adverse pregnancy outcome. An American

study involving 520,000 live births had found that new born of women with hyperemesis complicating pregnancy had significantly low birth weight and were likely to be Small for GA[128]. Contradictory results in previous studies may be due to heterogeneity in the methods, definition and multiple confounding factors. The main aim of our study is therefore to analyse association between hyperemesis and adverse pregnancy outcome. In a large cohort of norwegian mothers HG requiring hospitalisation were not associated with increased risk for preterm birth, LBW or SGA. Pregnancies complicated by HG had a slightly shorter length of gestation. There wasn't any difference in birth weight as per the maternal HG-status was concerned. Timepoint of hospitalisation had no influence on birthweight or age of gestation. Hyperemesis gravidarum was found to be associated with decreased risk of having Apgar score less than 7 at 1 minute, whereas there was no significant difference in risks for Apgar score less than 7 at 5 minutes [132].

Hyperemesis gravidarum:

Hyperemesis gravidarum is derived from a Greek word hyper- which means excessive and emesis meaning vomiting, and Latin word gravidarum, which is the feminine plural form of a adjective, which is here used as a noun, meaning "pregnant woman". Hence , hyperemesis gravidarum means "excessive vomiting of pregnant women".Hyperemesis gravidarum is seen in 1-3% of pregnant women[12]. Hyperemesis gravidarum is a condition in which diagnosis is by exclusion and is mostly based on the presentation. Definition of hyperemesis gravidarum in terms of clinical research is, persistent excessive vomiting,ketonuria and loss of weight > 5% of pre-pregnancy weight. There are other conditions in pregnancy with similar symptoms.

If there is no proper response to treatment or worsening of symptoms to such a extent that the patient has to be evaluated for other causes like acutegastroenteritis , pancreatitis , cholecystitis, peptic ulcer disease, hepatitis viral infestion , pyelonephritis and fatty liver of pregnancy[13]. Other possibilities of hyperemesis gravidarum could be due to toxicity of drugs, disorders in eating , gastroparesis, migraine headache , torsion of ovary , pseudotumorcerebri , psychosocial disorder and central nervous system tumours .

TYPE	• DIFFERENTIAL DIAGNOSIS
Gastrointestinal disorders	•acute appendicitis •acute cholecystitis • acute pancreatitis •Fatty infiltration of liver •Peptic ulcer • obstruction of small bowel
Infections	•Urinary tract infection •Hepatitis •Meningitis •Gastroenteritis
Metabolic	•Thyrotoxicosis (common in Asian subcontinent) •Addison disease •Diabetic ketoacidosis •Hyperparathyroidism •Vestibular disease
Drugs	• Antibiotics • drugs like Iron supplements
Gestational trophoblastic diseases	• Molar pregnancy • choriocarcinoma

Risk factors for HG:

In study published way back in 1987, showed that maternal factors like young age,nulliparity and obesity were associated with high risk of hyperemesis gravidarum[14]. Some fetal conditions like twin gestation, triploidy, gestational trophoblastic diseases, hydrops fetalis and down syndrome were also reported to be associated with increased risk of hyperemesis gravidarum. Women who had hyperemesis in their previous pregnancy had a high risk for recurrence in their following pregnancies[119,120].

Complications of Hyperemesis gravidarum:

Until 1950 excessive nausea and vomiting was found to be the cause of maternal death[16]. At present hyperemesis gravidarum is the 3rd leading cause of hospitalisation as a result of dehydration, electrolyte imbalance and malnutrition[17]. Currently maternal mortality due to hyperemesis is very rare. Wernicke's encephalopathy due to thiamine deficiency is a rare complication of hyperemesis gravidarum. It may result in symptoms such as ataxia, mental confusion and ophthalmoplegia[18]. Disorientation and inattentiveness is seen in some patients. It can also lead to stupor in patients followed by coma and eventually death patient is not treated appropriately. Few rare but lethal complications in the mother include acute kidney injury , pneumomediastinum, rhabdomyolysis, coagulopathy, sialorrhoea – constant salivation ("ice-cream bucket syndrome") and central pontine myelinolysis[19-24].Mallory-Weiss tears and esophageal rupture can also occur due to hyperemesis[121,122]. Hyperemesis gravidarum is found to be associated with high risk

of low birth weight[25].Attempts have been made to quantify symptoms of NVP resulted in Rhodes scale, which was originally designed for assessing the severity of nausea and vomiting in patients on chemotherapy for cancer[123].

In the year 2002, a scoring system called the Pregnancy-Unique Quantification of Emesis scoring system was proposed which was the first scale to focus on nausea and vomiting in pregnancy [124].in 2005 the 12-hour PUQE scoring system was validated [125].As per the Rhodes scale, the original PUQE scale assessed severity of NVP by mainly focusing on the number of hours of nausea and the number of episodes of retching and vomiting, as well as the overall well-being score in the 12 hours period just before assessment. Subsequently PUQE scale was revised to 24-hour scale taking into account the time spent for sleeping.

Motherisk PUQE-24 scoring system					
How long in the last 24 hours**, Have you felt nauseated or sick to your stomach?**	Not at all (1 pt)	1 hour or less (2 pts)	2-3 hours (3 pts)	4-6 hours (4 pts)	More than 6 hours (5 pts)
Have you vomited or thrown up in the last 24 hours?	7 or more times (5 pts)	5–6 times (4 pts)	3–4 times (3 pts)	1–2 times (2 pts)	I did not throw up (1 pt)
How many times did you have retching or dry heaves without bringing anything up in the last 24 hours?	No time (1 pt)	1–2 times (2 pts)	3–4 times (3 pts)	5–6 times (4 pts)	>=7 times (5 pts)
PUQE-24 Score: Mild = 6 score; Moderate = 7–12 score ; Severe = 13–15 score					

Investigations:

Initial laboratory investigations must include the following

- **Urine analysis for specific gravity and ketones**. Is sign of starvation; ketones may be harmful to fetal development. Specific gravity increases with depletion of volume.
- **Urine culture** – Urinary tract infection can cause nausea and vomiting and hence urine culture and sensitivity has to be done.
- **TSH and free thyroxine**: Hyperemesis gravidarum is seen in correlation with a transient hyperthyroidism and decreased TSH levels in 50-60% of cases. However, elevated levels free thyroxine suggests overt hyperthyroidism and necessitates further workup and management.
- **Serum electrolytes and ketones**: the electrolyte status has to be assessed for hypokalemia or hyponatremia and to identify hyperchloremic metabolic alkalosis or acidosis. Its also needed to evaluate renal function and to assess the volume status.
- **Hematocrit:** This test is done to look for haemoconcentration occurring as a result of overt dehydration.
- **Liver enzymes and bilirubin**: Increased transaminase levels may be seen in about 50% of patients with hyperemesis gravidarum. It usually resolves once nausea getssettled. Significant increase in liver enzymes, could be a sign of any other underlying liver conditions like viral hepatitis, ischemic hepatitis or autoimmune hepatitis, or any other liver injury warrenting specific treatment.

- **Serum Amylase and serum lipase:** Elevated Amylase level is seen in about 10% of patients with hyperemesis. Elevatd Lipase levels combined with an elevated amylase levels increases the specificity of diagnosing pancreatitis.

Treatment:

Early treatment of NVP decreases the risk of hyperemesis . There is no screening technique to predict the development of hyperemesis gravidarum in women with nausea and vomiting in pregnancy. There is no standardized treatment established for hyperemesis gravidarum.

Management of nausea and vomiting in pregnancy mainly depends on the severity of symptoms and its impact on the quality of woman's life and safety of the fetus. If the symptoms of nausea and vomiting does not improve with non pharmacologic interventions drugs are prescribed. Adequate and appropriate treatment involves balance between non pharmacologic and pharmacologic methods.

Nonpharmacologic interventions:

The first line of treatment of NVP in pregnancy involves diet changes and life style modifications. Though harmless, there is no evidence to support its efficacy of this method as a treatment for HG.

Diet:

Symptoms can be relieved by modifying the quantity and size of meals that is consumed the whole day. Frequent small meals and fluid intake at regular intervals can prevent nausea and vomiting becoming worse. These are advised to take bland

diet and to avoid strongly odorous foods. Meals should be rich in carbohydrates and protein and should contain less amount of fat. Light snacks like nuts, dry fruits and beans are often advised in the diet. Foods that trigger nausea should be avoided and it varies among each women. Prenatal vitamins at night and discontinuation of oral iron therapy are found to be helpful in women with nausea or vomiting in pregnancy[36]. Most women complain of increased sensitivity to smell which leads to worsening of symptoms. Triggering factors which has to be avoided in women with NVP are certain odour like perfume, food smell ,very loud noise and pressure over the abdomen.

Lifestyle Modification:

In women affected by hyperemesis gravidarum stress should be avoided and should take enough rest. Supportive counselling may be necessary**. L**ifestyle modifications include napping quiet often and avoiding certain stimuli which provokes nausea and vomiting. Sleep requirements increase in pregnancy, therefore patients should be encouraged to have adequate sleep because fatigability can exacerbate nausea and vomiting.

Liberal encouragement on diet and lifestyle changes has been resommended by the Society of Obstetricians and Gynaecologists of Canada and it also recommends that women should be counselled to eat whatever is appealing to them to soothen their symptoms[47].

Pharmacological Therapy.

Pregnant women are usually not included in most of the drug trials. Therefore there is not much evidence available regarding the efficacy and safety of drugs in

pregnancy. At present there is no drug which is approved by FDA for treating nausea and vomiting and no treatment protocol has been established.

Few well controlled studies have found that the FDA class A drugs are not found to be associated with increased risk of fetal anomalies . Whereas Class B drugs have not caused any fetal risk in animal studies without any controlled studies in pregnant women, or few animal studies have shown an adverse effect that has not been seen or confirmed in controlled studies involving women who are pregnant. Drug belonging to classes D and X have been shown to have teratogenic side effects..

Whereas class C drugs, there is little or no evidence regarding the safety of these drugs. But class C drugs are prescribed in pregnancy if the benefit overweighs the fetal risk. As per the Centres for Disease Control and Prevention, the rate of congenital anomalies is approx 3% according to drug safety studies[48].

Multivitamin Supplementation:

THIAMINE:

Daily intake of thiamine should be 1.5mg/day among pregnant women If not possible orally it should be taken as in infusion at a dose of 100mg in 100 ml normal saline over 30 mins weekly..Two studies have been found to support multivitamin supplementation for prevention of nausea or vomiting. A study was conducted in 301 pregnant women to analyse the relation between nausea and vomiting and multivitamin supplementation.[49] Vomiting was seen in patients with a lack of multivitamin supplementation at less than 6 weeks of' gestation, during which the women is unaware of their pregnancy. One more study compared t vitamin

supplementation with placebo during the periconceptional period and was found that vitamin supplementation was associated with significantly reduced incidence of nausea and vomiting.[50] In this particular study, women were asked to take a daily multivitamin supplementation for at least 1 month before conception and asked to continue throughout the 1st trimester.

It is beleived that positive effect of multinitamin was due to the balance in the nutritional status and basal metabolism.biologic basis of the association between decreased vitamin supplementation and increased risk of hyperemesis has to be evaluatrd throygh more studies.. All women in reproductive age group should be prescribed daily ivitamin and folic acid for their general health and to reduce the risk of neural tube defects..

Pyridoxine:

Pyridoxine (vitamin B_6) is a water-soluble vitamin and is an important coenzyme in the lipid metabolism , amino acids and carbohydrates metabolism. It can be given alone or can be given with doxylamine for treating of nausea and vomiting. When it is given alone, the dosage should be 25 mg of tab pyridoxine every 8 hourly or 75 mg/day.[52] One more study showed that there is no correlation between nausea and vomiting and low levels of pyridoxine.[51]Some studies have found it to be effective in the management of nausea and vomiting in pregnancy.[52,53]

The safe use of pyridoxine in combination with doxylamine has been extensively studied.[54] A retrospective study on pyridoxine monotherapy showed that

no significant risk of major congenital malformations in 458 cases when compared to 911 controls (relative risk 1.05).[55]

Pyridoxine as single agent has been studied in only 2 randomized trials.[52,53] A random control trial was conducted to assess the efficiency of 25 mg of pyridoxine given every 8th hourly for 3 days and compared with placebo to manage nausea and vomiting.[52] The analysts found only mild difference among women with mild to moderate symptoms. In women with severe nausea a significant improvement was seen .A statistically significant reduction in the number of episodes of vomiting was seen in these patients on pyridoxine .Another randomized, double-blind trial was conducted to assess the efficacy of increased dose of 30mg pyridoxine /day Vs placebo for 5 days.[53] There was a significant decrease in nausea and vomiting, with a decreasing trend in the number of episodes in the test group when compared with control group. Pyridoxine has an excellent safety profile with very less side effects . Therefore, it is an acceptable first-line drug for nausea and vomiting either given alone or in combination with drugs like doxylamine.

Doxylamine-Pyridoxine.

The dose recommended is doxylamine 10 mg and 10 mg pyridoxine. Upto 4 tablets can be given per day in patients who have severe nausea or vomiting[57].

A large number of case control and cohort studies have been done and it showed that doxylamine pyridoxine combination was very safe in pregnancy and had no adverse side effects on the fetus.[54] Several other randomized control trials also

showed that doxylamine-pyridoxine combination was effective in reducing nausea and vomiting in pregnancy.[61-63]

Antihistamines:

The first line anti emetic drugs recommended by the American Congress of Obstetricians and Gynecologists as per the 2004 guidelines on nausea and vomiting in pregnancy are metaclopramide , dimenhydrinate and promethazine.[1]

A large number of dopamine antagonists are used in the treatment of nausea and vomiting in pregnancy. They act by antagonizing the action of dopamine with D2 receptors by exerting a antiemetic effect. There are 3 main classe of dopamine receptor antagonists, they are phenothiazines ,butyrophenones and, benzamides. Phenothiazines used are prochlorperazine and promethazine, whereas metaclopromide is a benzamide.

There is no standard treatment protocol for treatment of nausea and vomiting in pregnancy internationally. Metaclopromide was the most commonly used drug in the south and western part of Europe. However phenothiazines and metaclopromide are second line drugs in the treatment of NVP because of its limited information on the safety of these drugs when given in pregnancy.

Serotonin Antagonists.

Serotonin antagonists exert their action at the 5-hydroxy-tryptamine$_3$ (5-HT$_3$)-receptors by acting centrally and peripherally. There is a generalised block at the level of small bowel, the vagus nerve, and the chemoreceptor trigger zone, thereby resulting

in reduced stimulation of the vomiting center in the medulla. Only a limited data is available regarding the safety of ondansetron in the treatment of nausea and vomiting in pregnancy

Furthermore studies are needed to establish the safety factor and efficiency of serotonin antagonists in the management of hyperemesis in pregnancy. It is given only in cases of refractory nausea and hyperemesis when all the above described methods have failed..

Corticosteroids:

A accurate regimen is not yet established for corticosteroids in the treatment of nausea and vomiting. One is a oral regimen and other is IV methylprednisolone 48mg per day in three divided doses for two to three days. If there is no response in 3 days the treatment has to be discontinued because improvement beyond 72 hours is unlikely. Otherwise dosage can be tapered over two weeks. For those with refractory vomiting lowest effective dose can be continued for 6 weeks but not beyond that due to side effects in the mother. Safety factor regarding management with corticosteroids in pregnant women is still controversial. A large meta analysis of studies showed a high risk of malformations after exposure to corticosteroids in the 1st trimester.[93] there was an 3.4 fold increased risk of developing oral clefts in fetus in those women receiving corticosteroids.[94,95] The overall prevalence of teratogenic side effects is 1 to 2 cases per 1000 treated women.[96]

It is better to avoid Corticosteroid therapy during the 1st trimester due to the possibility of high risk of cleft lip formation in fetus. Corticosteroids are reserved for treatment of recurrent or refractory NVP or hyperemesis gravidarum.

Alternative and complementary therapies :

Other traditional measures used in the treatment of nausea and vomiting in pregnancy are ginger and accpunture. Other alternative therapies are chamomile, peppermint and raspberry leaf.[98]

Ginger:

The gastrointestinal symptoms of morning sickness and hyperemesis gravidarum is similar, therefore, the ginger root called as ***Zingiber officinale***, has been shown to treat hyperemesis. For many years ginger has been used in treating nausea and vomiting in pregnancy. It is available in various forms like tablets, tea and ginger root has been consumed directly. Its effectiveness is said to be mainly due to its aroma, carminative and absorbent properties.[104] The commonest side effect associated with ginger is bloating , belching and it can leave a bad taste in the mouth. Patients with bleeding disorders and those taking aspirin or heparin should use ginger with caution because ginger may also inhibit platelet aggregation. Few studies have compared ginger with placebo and found that those patients who took ginger had a significant reduction in nausea and vomiting. Similarly another study compared ginger and pyridoxine and found that both were equivalent in the management of nausea and vomiting in pregnancy. The advantage of ginger is that it is very inexpensive and

easily available. But further research is needed to know the efficacy and safety of using ginger in women who are pregnant.

Acupressure Treatment:

Acupressure is nothing but the stimulation by using pressure on certain sites called as acupoints. The commonly used acupoint in treating NVP is pericardium 6 (P6) or a point called Neiguan point situated 3 finger width above the wrist joint on the inner surface of the arm. The main advantage is there is no outside source of risk to the fetus.[112] Ot her possible mechanisms of action for decreasing hyperemesis from acupuncture are that it inhibits nociceptive transmission and autonomic reflexes. It decreases pain in system from the periaqueductal gray matter , which works by endorphinergic mechanisms. Accupuncture also acts on the Gastrintestinal tract thereby improving the gastric emptying , lack of which is one of the reason for hyperemesis in pregnancy, another mechanism of action is through somatovisceral reflexes.[138]

The Technique of Hypnosis:

Hypnosis is a method used to control physiologic changes that are said to be involuntary. The sympathetic tone, muscle tone, vasodilatation heart rate and vasoconstriction are said to be controlled by the hypnotised patients [7].It is compared with biological feedback in which the patients are taught to voluntarily control the above said mechanisms. External method of feedback is used by biofeedback whereas internal control from the patient is used in hypnosis.

Dissociation of content is the mechanism through which it acts, the patients attention is focused on one particular task and rest of the task surrounding them are masked or is made temporarily not reachable. Hypnosis might also act through dissociation of context, in which this narrowing of attention briefly involves higher order processes.

It is, however, important to dispel myths or doubts that the patients have about hypnotic treatment. There are no teratogenic effects noted.[7] It has also been found that providing this treatment to patients with morning sickness can prevent nausea and vomiting from becoming worse as well as prevents the disease progression to hyperemesis gravidarum..

Ideally the initial and foremost management for hyperemesis gravidarum includes intravenous fluid administration, vitamin supplementation ,electrolyte replacement, and prescribing antiemetic drugs..[13]

RESULTS

AGE DISTRIBUTION AMONG TEST GROUP AND CONTROL GROUP

TABLE 1

AGE	TEST	CONTROL
20-25 YRS	41[51.25%]	40 [50%]
26-30 YRS	26[32.5%]	19 [23.75%]
31-35 YRS	11[13.75%]	18 [22.5%]
36-40 YRS	2[2.5%]	3 [3.75%]
TOTAL	**80**	**80**

The age distribution was almost similar both the study group and the control group. Almost 50% of the participants were in the age group of 20-25 yrs.

AGE DISTRIBUTION AMONG CASES AND CONTROL

CHART 1

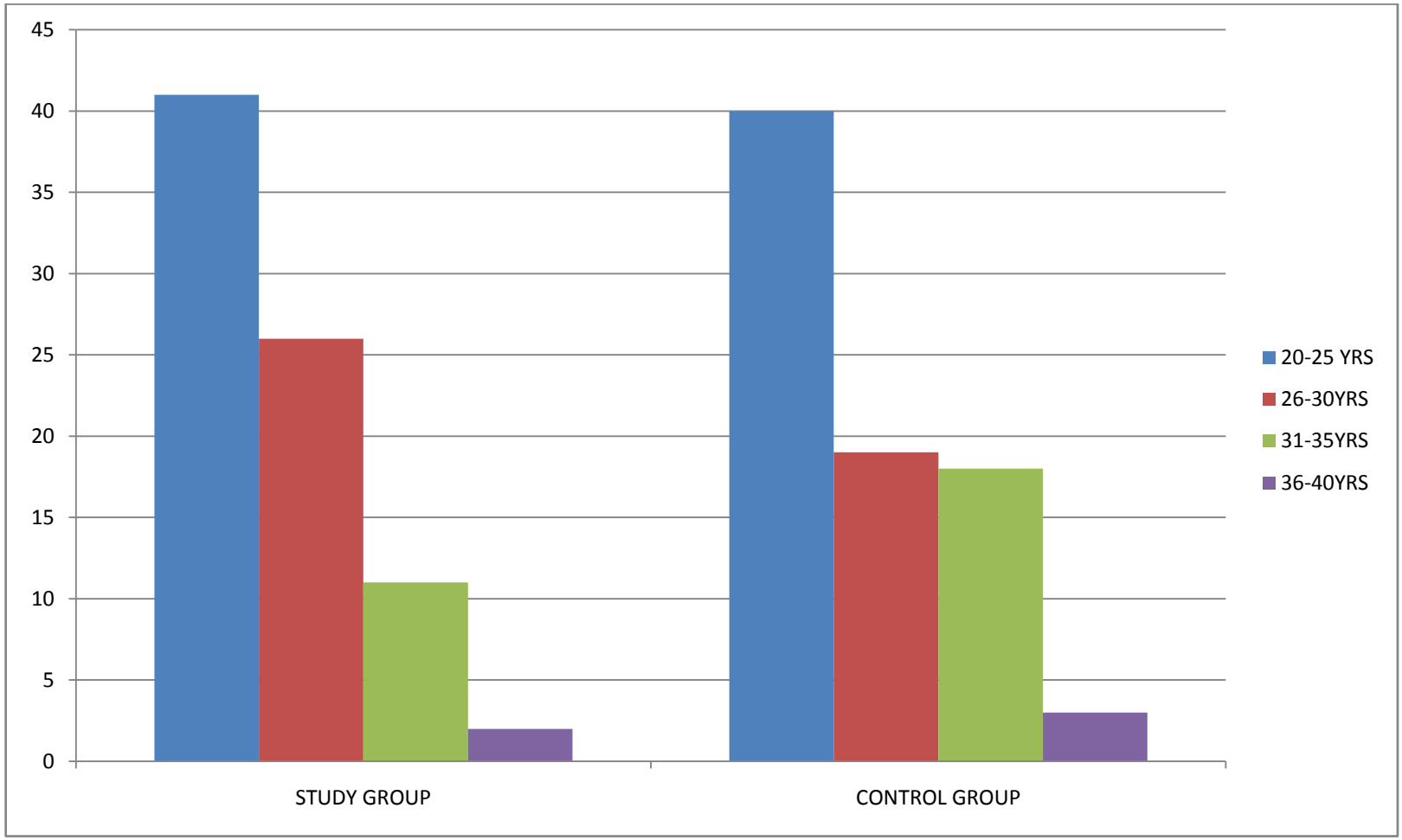


TABLE 2

PARITY DISTRIBUTION AMONG STUDY GROUP AND CONTROL GROUP

PARITY	STUDY GROUP	CONTROL GROUP
PRIMIGRAVIDA	52	55
MULTIGRAVIDA	28	25
TOTAL	80	80

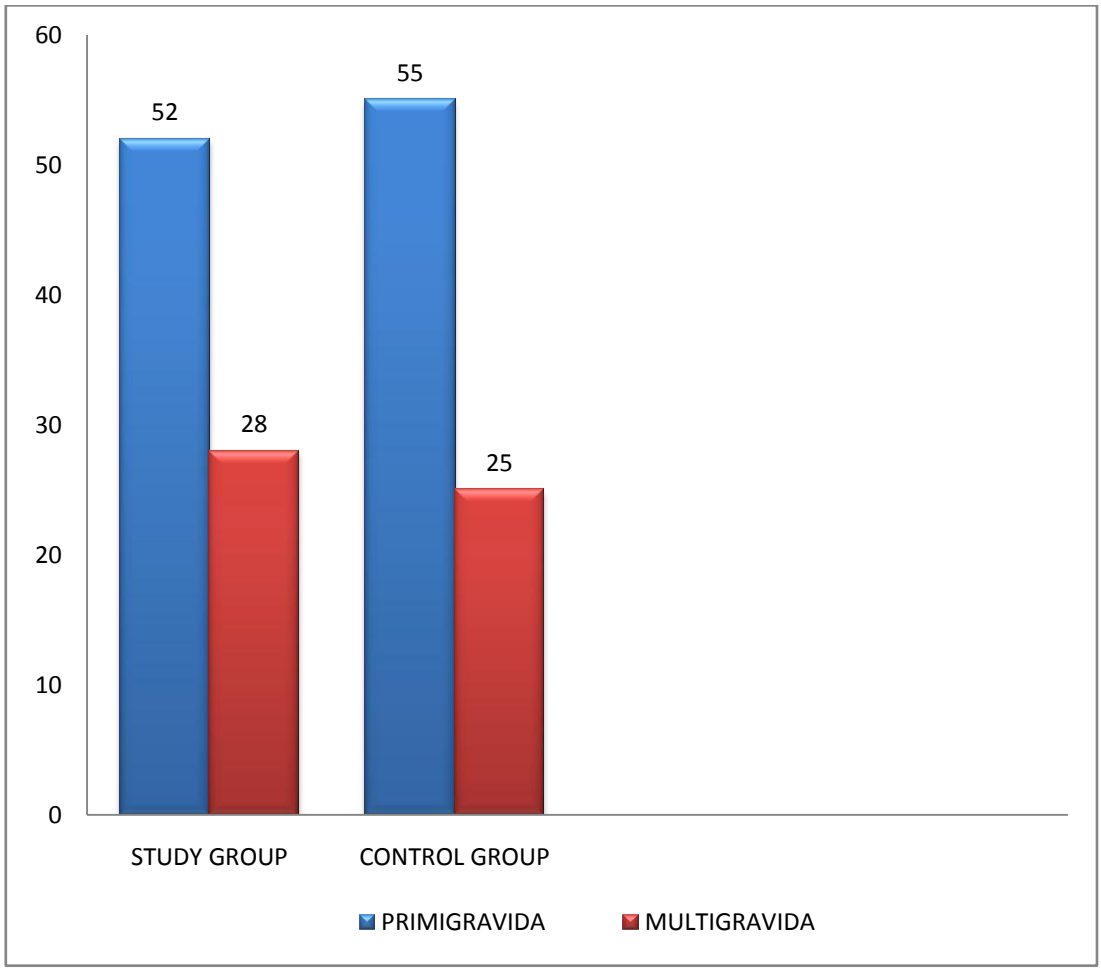
% of Primigravida in Study group : 65%

% of Multigravida in Study group: 35%

% of Primigravida in Control group: 68.75%

% of Multigravida in Control group: 31.25%

CHART 2



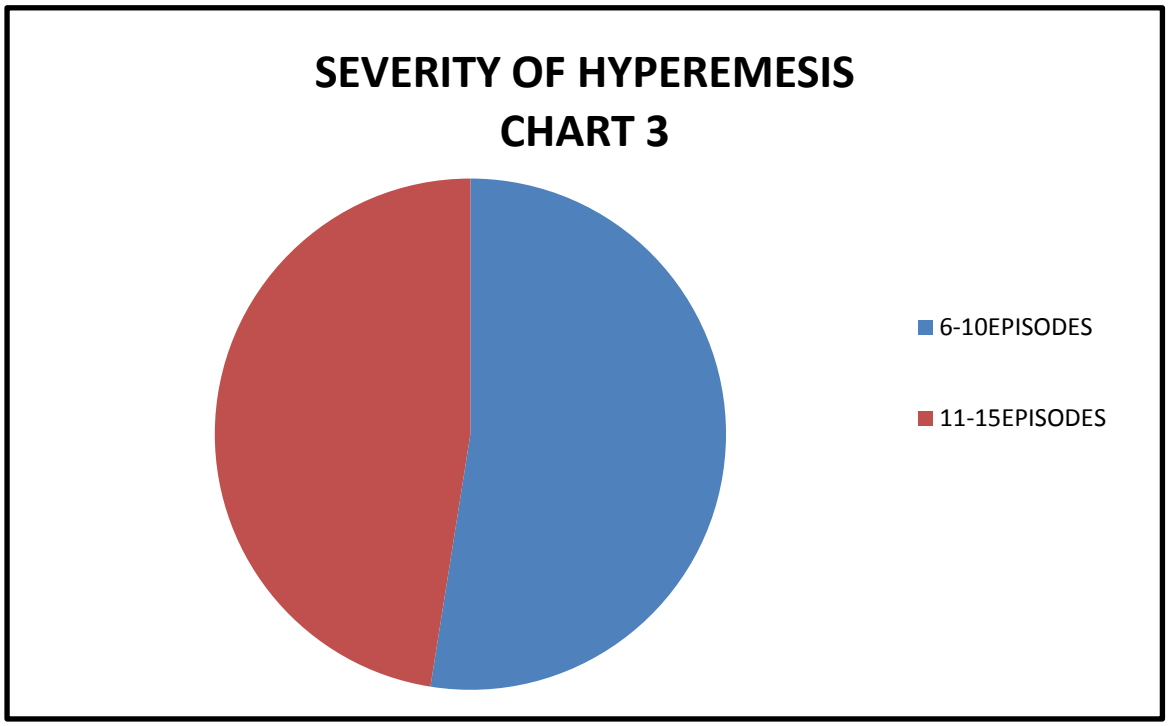
The distribution of primigravida and multigravida was almost same among the study group and the control group.

SEVERITY OF HYPEREMESIS

TABLE 3

NO OF EPISODES	SAMPLE
6-10 EPISODES	42
11-15 EPISODES	38

CHART 3



PREVALENCE OF ANAEMIA AMONG TEST AND CONTROL GROUP

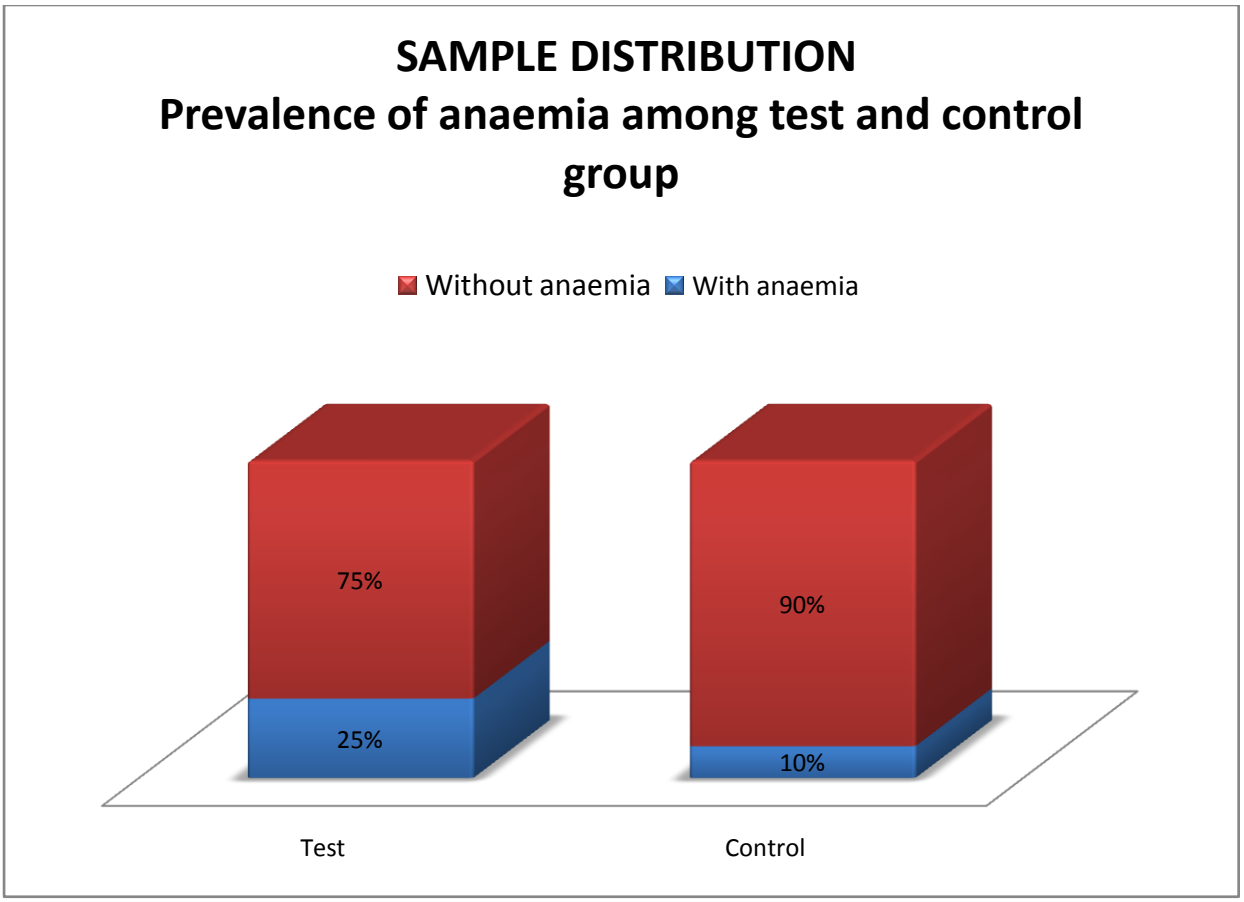
TABLE 4

ANEMIA	STUDY GROUP	CONTROL GROUP	TOTAL	CHI SQUARE VALUE	P VALUE
YES	20	8	28	6.234	0.013
NO	60	72	132		

Prevalence of anemiain test group - 25 %

Prevalence of anemia in control group – 10%

CHART 4



There was a significantly increased prevalence of anemia in patients in whom pregnancy was complicated by hyperemesis gravidarum , as indicated by the p value

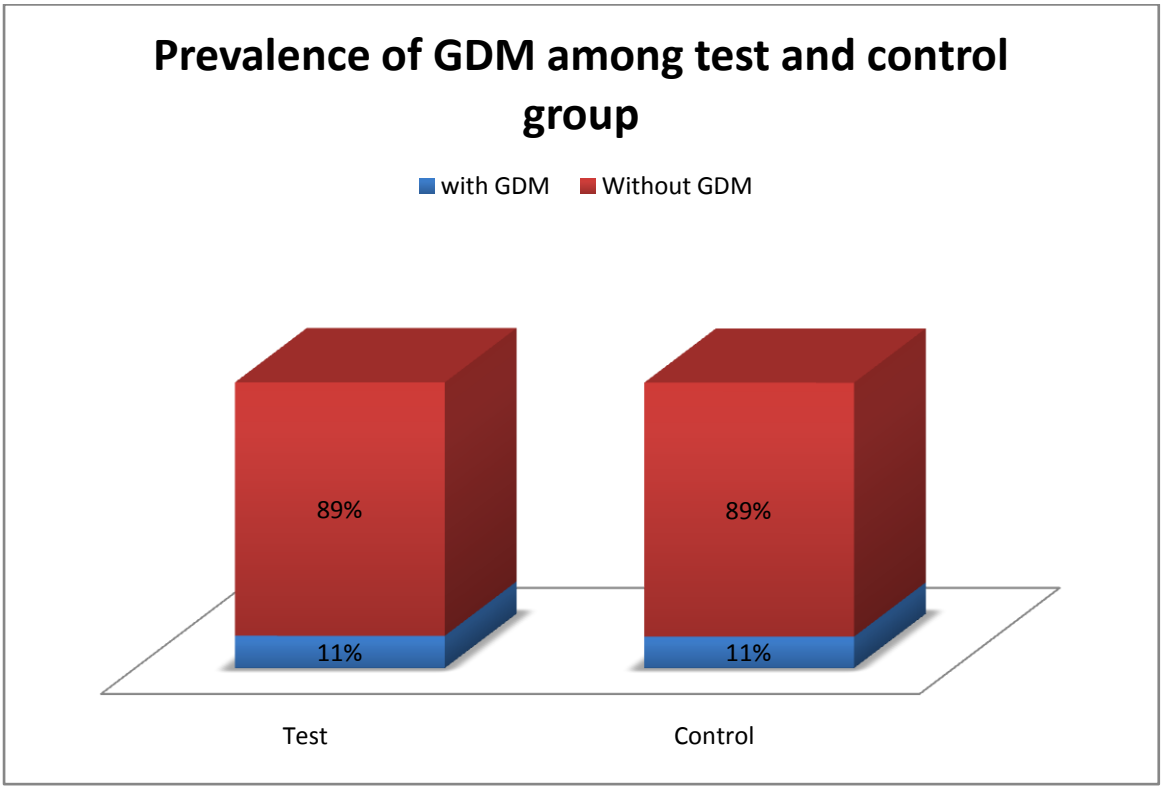
PREVALENCE OF GDM AMONG TEST AND CONTROL GROUP

TABLE 5

GESTATIONAL DIABETES MELLITUS	STUDY GROUP	CONTROL GROUP	CHI SQUARE VALUE	P VALUE
YES	9	9	.000	1.000
NO	71	71		

In my study the prevalence of gestational diabetes mellitus was same among both study group and the control group

CHART 5



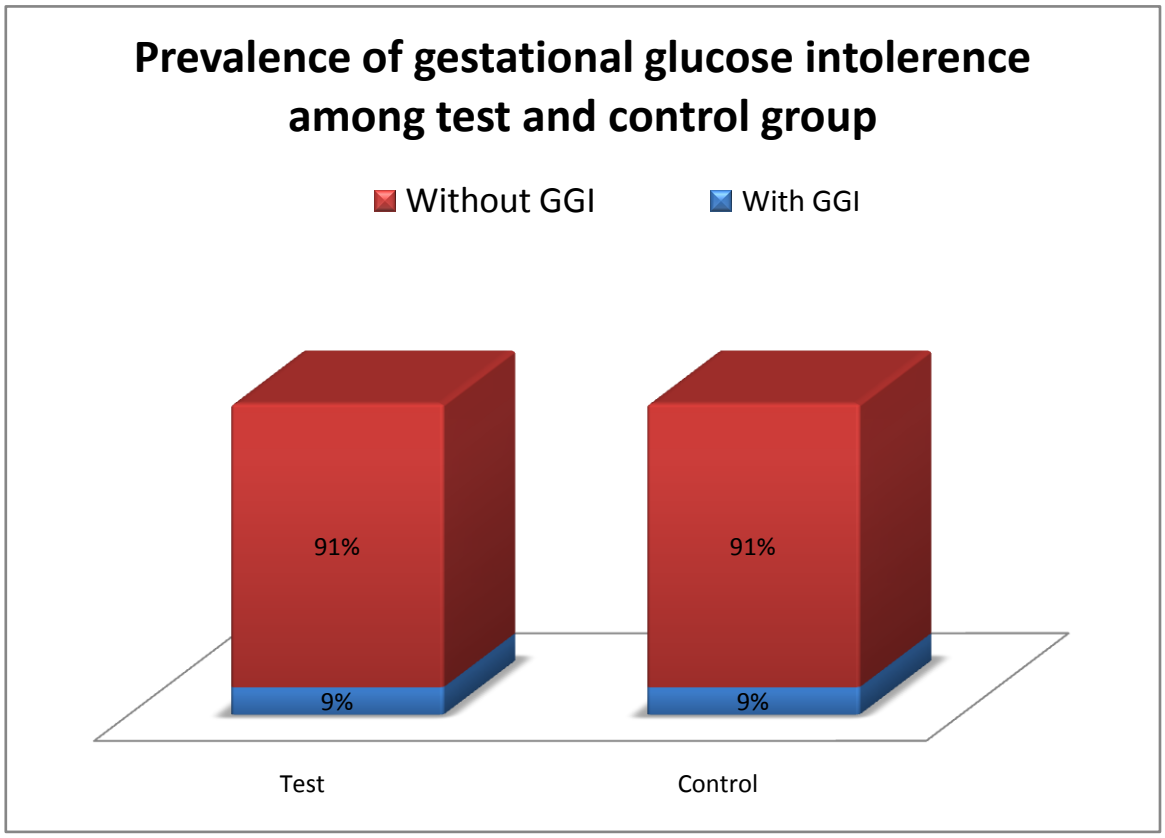
PREVALENCE OF GESTATIONAL GLUCOSE INTOLERANCE AMONG CASES AND CONTROL

TABLE 6

GESTATIONAL GLUCOSE INTOLERNCE	STUDY GROUP	CONTROL GROUP	CHI SQUARE VALUE	P VALUE
YES	7	73	.000	1.000
NO	7	73		

The prevalence of gestational glucose intolerance was 9% in both study group and control group, which was insignificant as indicated by the p value.

CHART 6



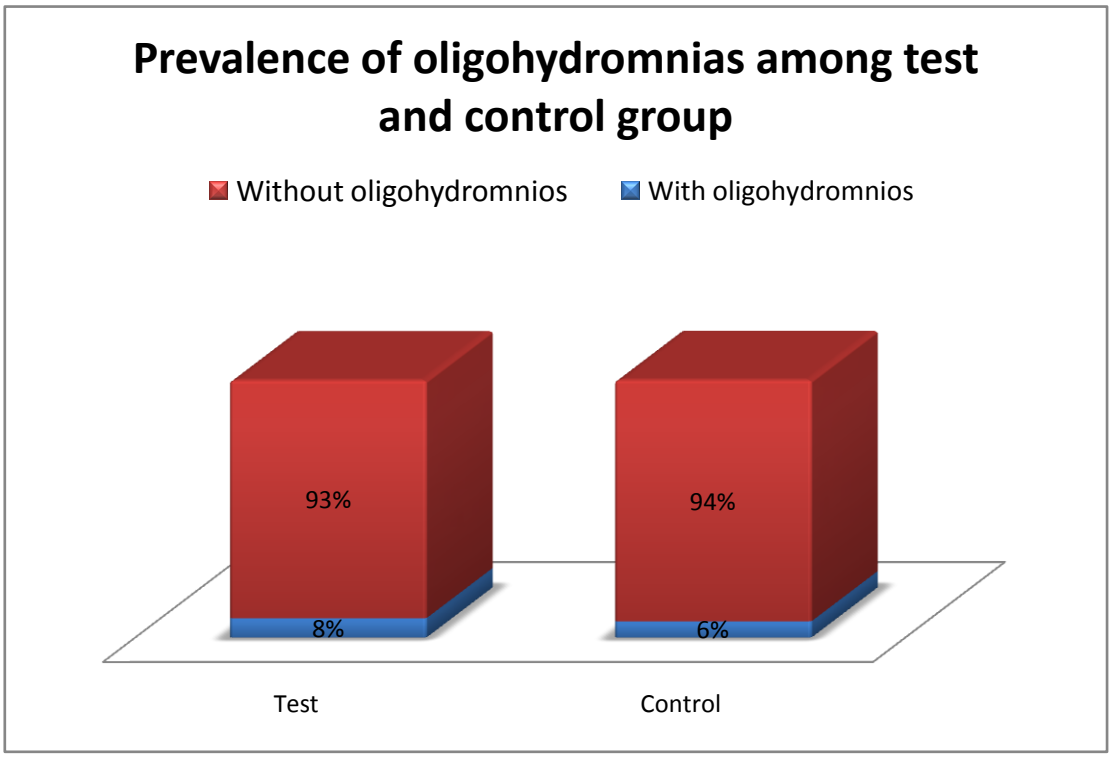
PREVALENCE OF OLIGOHYDRAMNIOS AMONG TEST AND CONTROL GROUP

TABLE 7

OLIGOHYDRAMNIOS	STUDY GROUP	CONTROL GROUP	CHI SQUARE VALUE	P VALUE
YES	6	5	.098	.755
NO	74	75		

The prevalence of oligohydramnios was also almost same among cases and control therefore was found to be insignificant as indicated by the p value

CHART 7



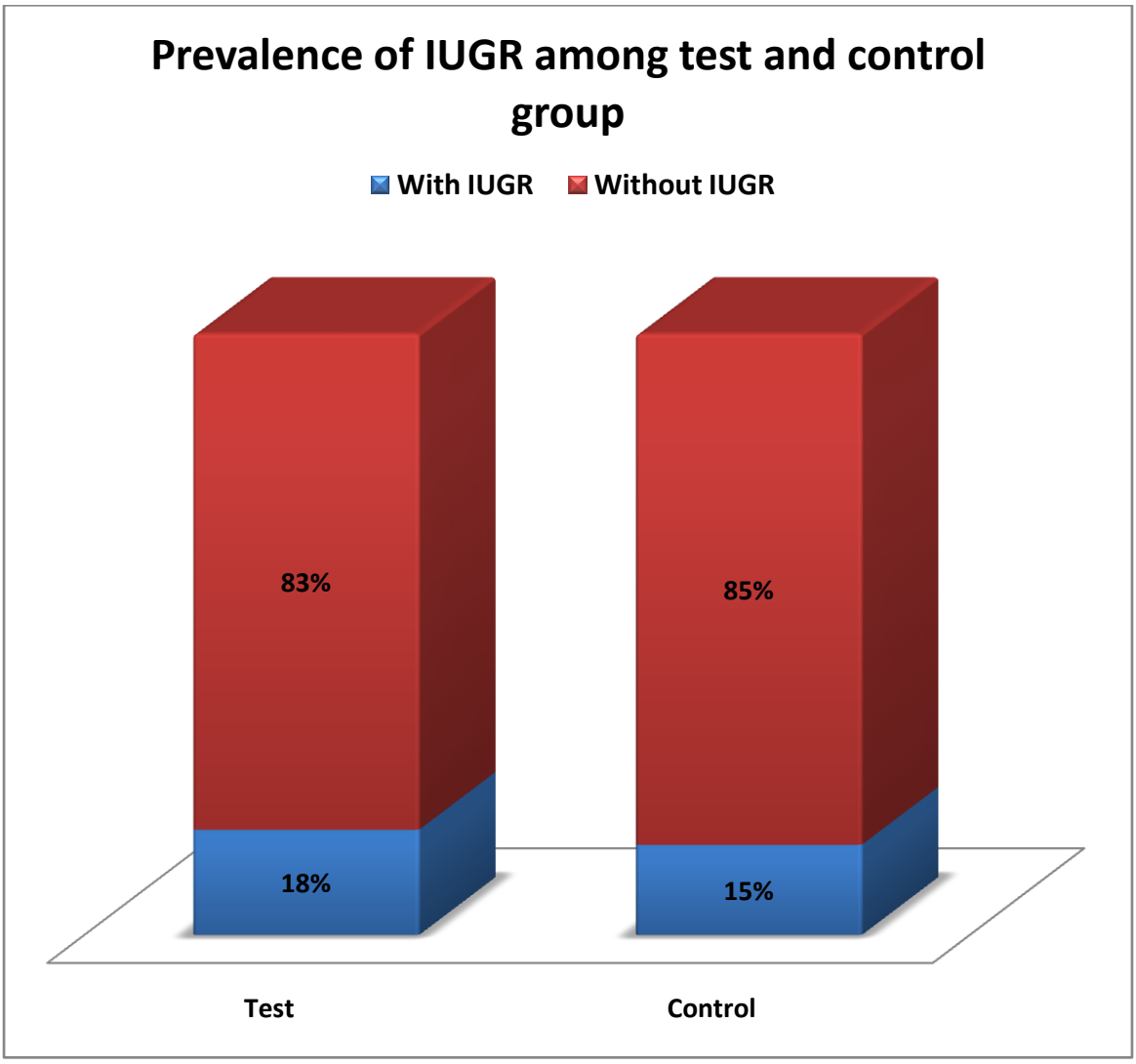
PREVALENCE OF IUGR AMONG CASES AND CONTROL

TABLE 8

IUGR	STUDY GROUP	CONTROL GROUP	CHI SQUARE VALUE	P VALUE
YES	14	12	0.184	.668
NO	66	68		

Out of 80 patients with hyperemesis gravidarum 14 patients had intrauterine growth restriction. Whereas in the control group 12 patients had intrauterine growth restriction.

CHART 8


Prevalence of IUGR among test and control group
With IUGR
Without IUGR
83%
18%
85%
15%
Test
Control

WEIGHT GAIN IN PREGNANCY AMONG STUDY GROUP AND CONTROL GROUP

TABLE 9

WEIGHT GAIN	NO WEIGHT GAIN	3-5kgs	6-8 kgs	9-11 kgs	12-14 kgs	15-18 kgs	19-21 kgs
STUDY GROUP	2	5	30	28	14	0	1
CONTROL GROUP	0	0	8	34	37	1	0
TOTAL	2	5	36	64	51	1	1

In my study out of 180 patients about 40% gained weight of about 9-11 kgs.

In my study group out of 80 patients with hyperemesis gravidarum about 30 patients (37.5%) gained weight of about only 6-8 kgs.

In the control group about 37 patients (46.3 %) gained weight of about 12-14 kgs.

In study group

Weight gain between 6-8kgs – 37.5%

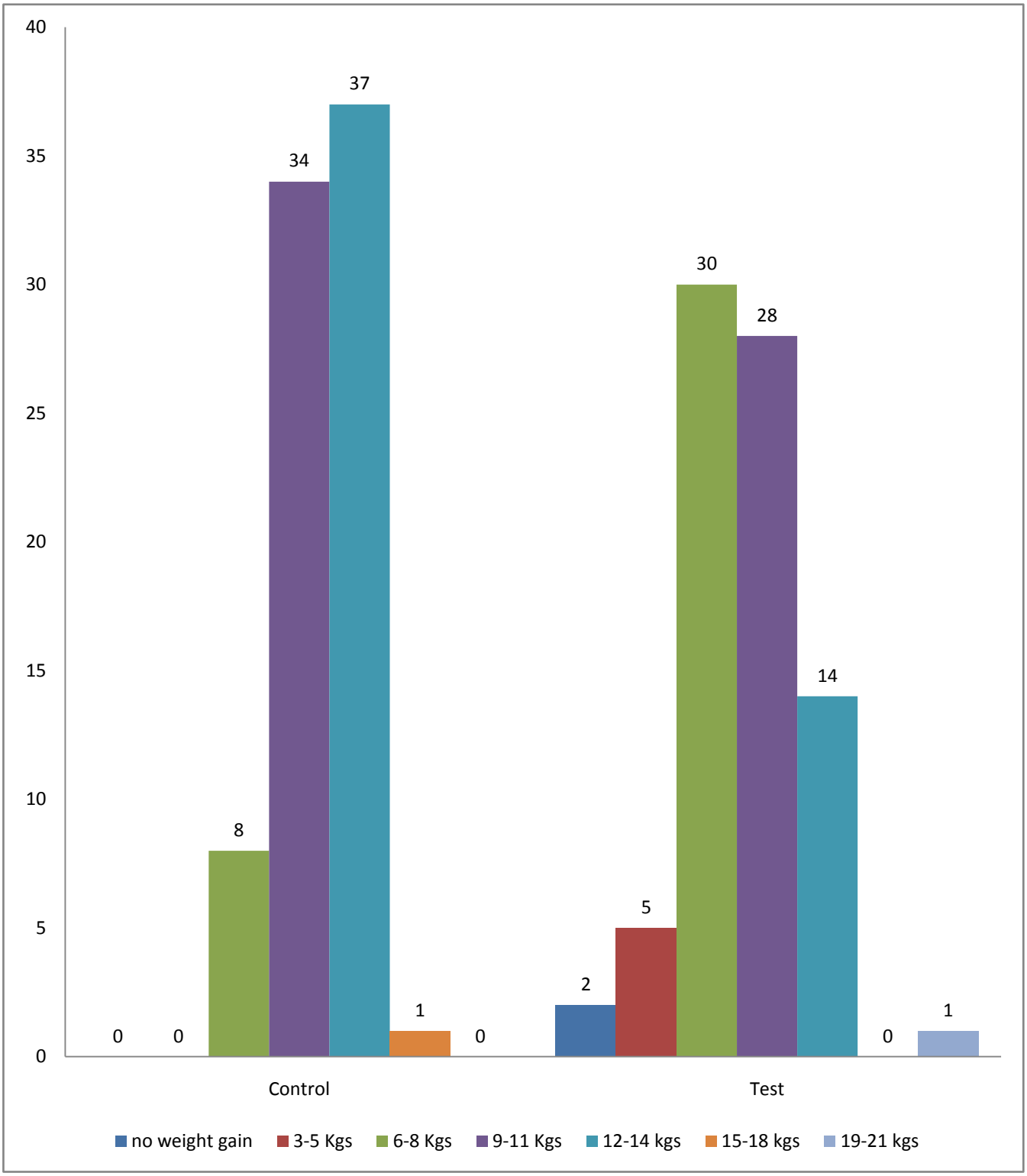
Weight gain between 9-11kgs – 35%

In control group

Weight gain between 12-14 kgs – 46.3%

Weight gain between 9-11kgs – 42.5 %

CHART 9


40
35
30
25
20
15
10
5
0
0
0
8
34
37
1
0
2
5
30
28
14
0
1
Control
Test
no weight gain
3-5 Kgs
6-8 Kgs
9-11 Kgs
12-14 kgs
15-18 kgs
19-21 kgs

GESTTIONAL AGE OF DELIVERY AMONG STUDY GROUP AND CONTROL GROUP

TABLE 10

TIME OF DELIVERY	ABORTION	PRETERM	TERM
STUDY GROUP	3	5	72
CONTROL GROUP	0	2	78
TOTAL	3	7	150

STUDY GROUP:

% of term delivery - 90%

% of preterm delivery - 6.3%

% of abortion - 3.8%

CONTROL GROUP

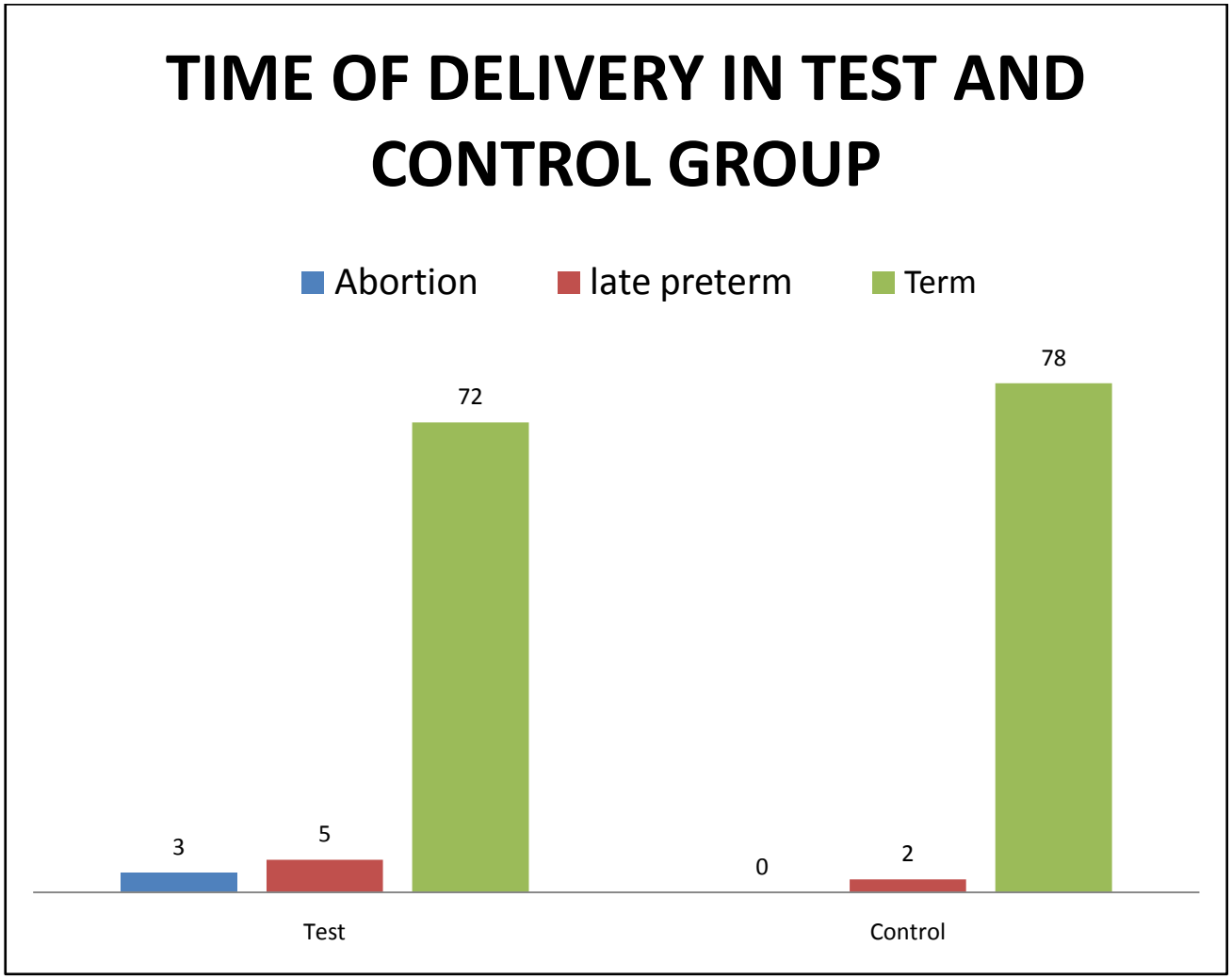
% of term delivery – 97.5%

% of preterm delivery – 2.5%

No abortions

P value – 0.158

CHART 10


TIME OF DELIVERY IN TEST AND CONTROL GROUP
Abortion
late preterm
Term
3
5
72
0
2
78
Test
Control

BIRTH WEIGHT DISTRIBUTION AMONG STUDY GROUP AND CONTROL GROUP

Table 11

BIRTH WEIGHT	1.5-2kgs	2.1-2.5kgs	2.6-3kgs	3.1-3.5kgs	3.6-4 kgs
STUDY GROUP	3	27	24	19	4
CONTROL GROUP	1	20	35	20	4

STUDY GROUP:

33.75% of babies had a low birth weight between 2.1 – 2.5 kgs

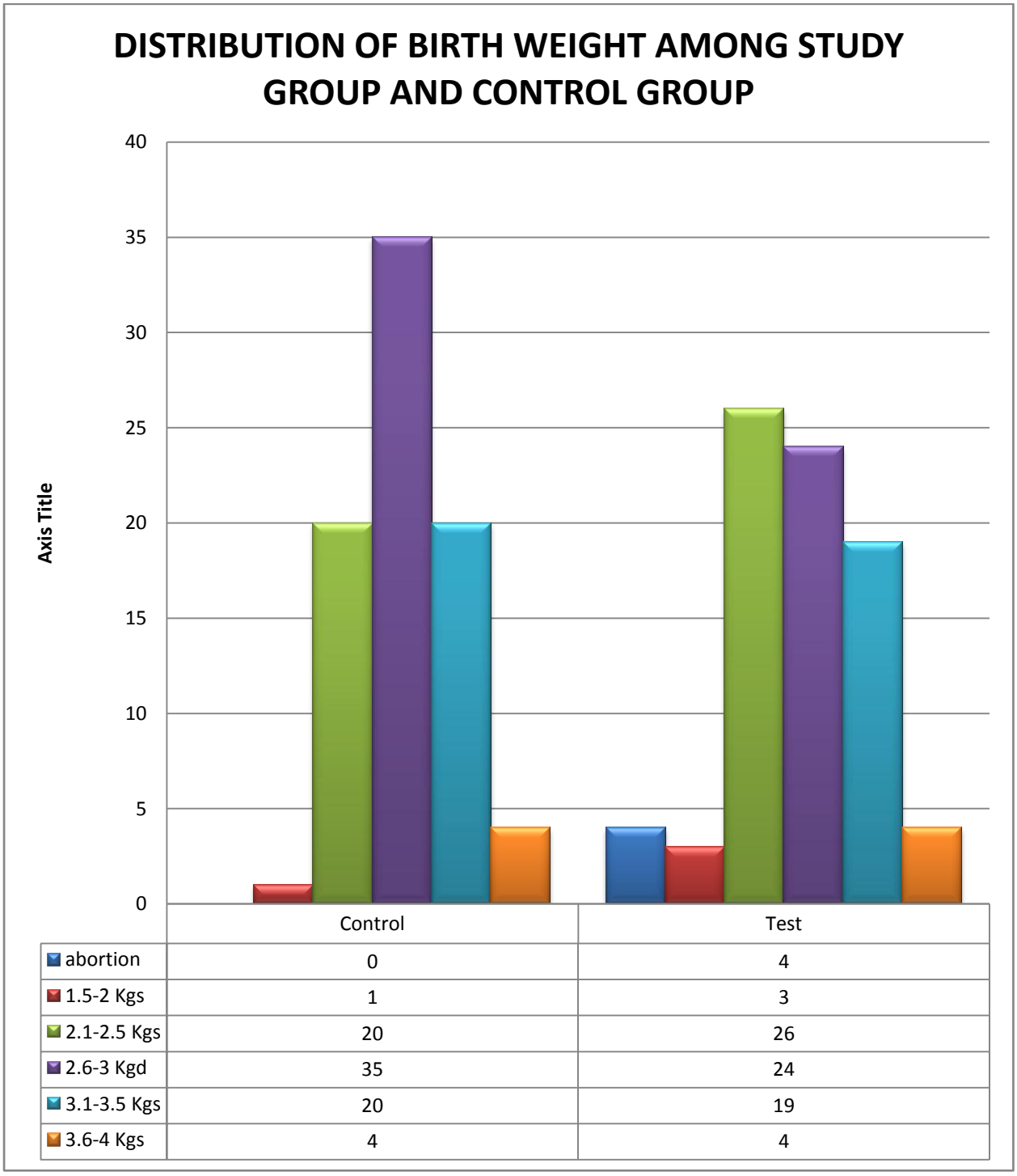
30% of babies had a birth weight between 2.6 – 3 kgs

CONTROL GROUP:

43.8% of babies had a birth weight between 2.6-3 kgs

25% of babies had birth weight between 3.1 – 3.5 kgs

CHART 11



	Control	Test
abortion	0	4
1.5-2 Kgs	1	3
2.1-2.5 Kgs	20	26
2.6-3 Kgd	35	24
3.1-3.5 Kgs	20	19
3.6-4 Kgs	4	4

DURATION OF HYPEREMESIS AND ITS EFFECT ON MATERNAL AND FETAL OUTCOME

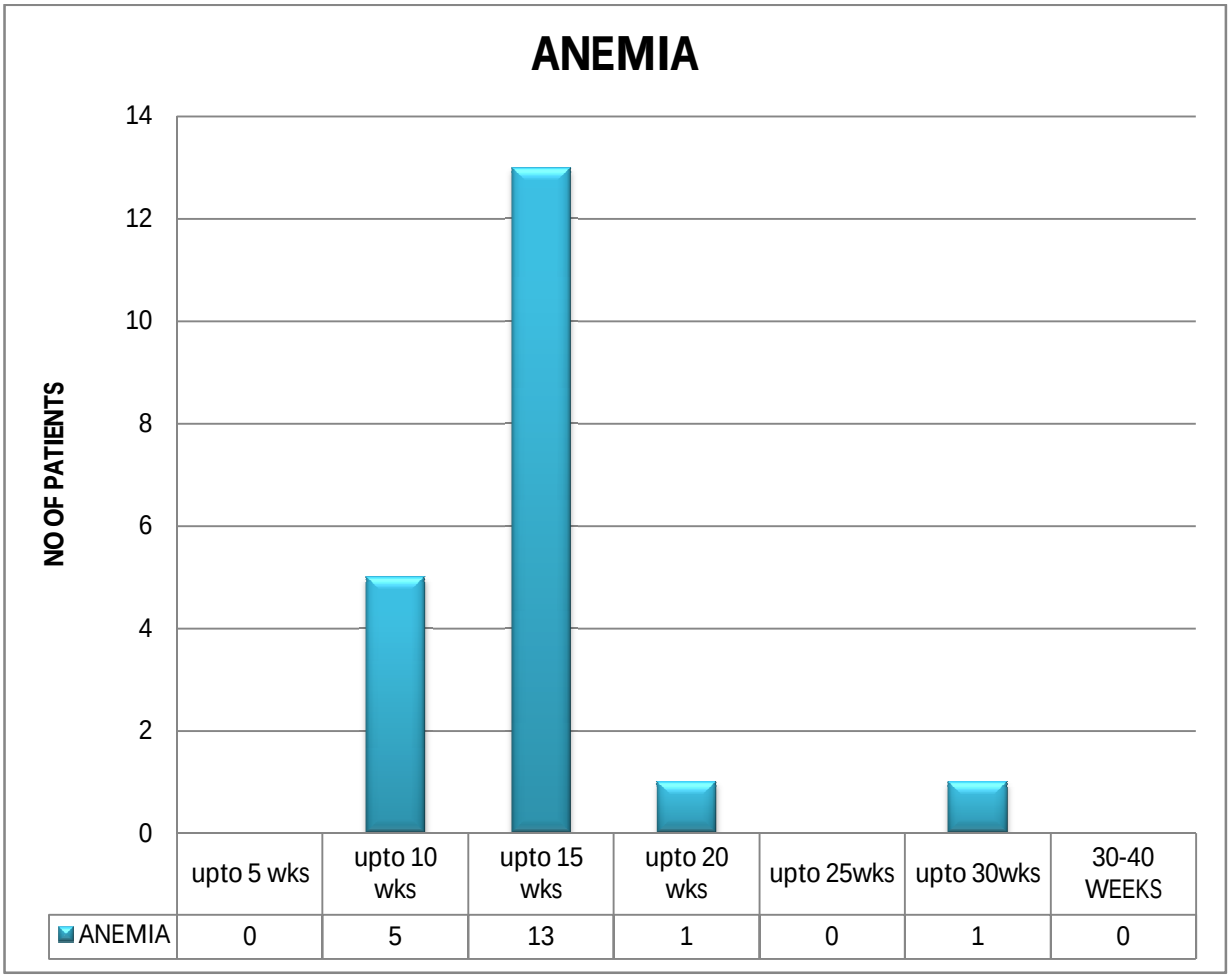
TABLE 12

GESTATIONAL AGE	ANEMIA
Upto 5 wks	0
Upto 10 wks	5
Upto 15 wks	13
Upto 20 wks	1
Upto 30 wks	1
TOTAL	20

Of 20 patients with hyperemesis gravidarum who developed anemia 13 patients were hospitalized for hyperemesis at 11-15 weeks. The incidence of anemia was more in the patients who had hyperemesis at later gestational age than those who had hyperemesis at less than 10 weeks.

CORRELATION BETWEEN DURATION OF HYPEREMESIS GRAVIDARUM AND ANEMIA

CHART 12



CORRELATION BETWEEN DURATIONOF HYPEREMESIS AND GESTATIONAL HYPERTENSION

TABLE 13

GESTATIONAL AGE	HYPERTENSION
Upto 5 WEEKS	0
Upto 10 WEEKS	1
Upto 15 WEEKS	2
Upto 20WEEKS	0
Upto 30 WEEKS	0
Upto 40 WEEKS	0
TOTAL	**3**

Only 3.8% of patients with hyperemesis gravidarum developed gestational hypertension of which 2.5% was seen in those patients who had hyperemesis between 11-15 weeks.

CORRELATION BETWEEN DURATION OF HYPEREMESIS AND GESTATIONAL DIABETES MELLITUS

TABLE 14

GESTATIONAL AGE	GESTATIONAL DIABETES MELLITUS
Upto 5 WEEKS	0
Upto 10 WEEKS	1
Upto 15 WEEKS	8
Upto 20WEEKS	0
Upto 30 WEEKS	0
Upto 40 WEEKS	0
TOTAL	**9**

11.3% of patients developed gestational diabetes mellitus of which 10% was seen in those patients who had hyperemesis between 11-15 weeks of gestation

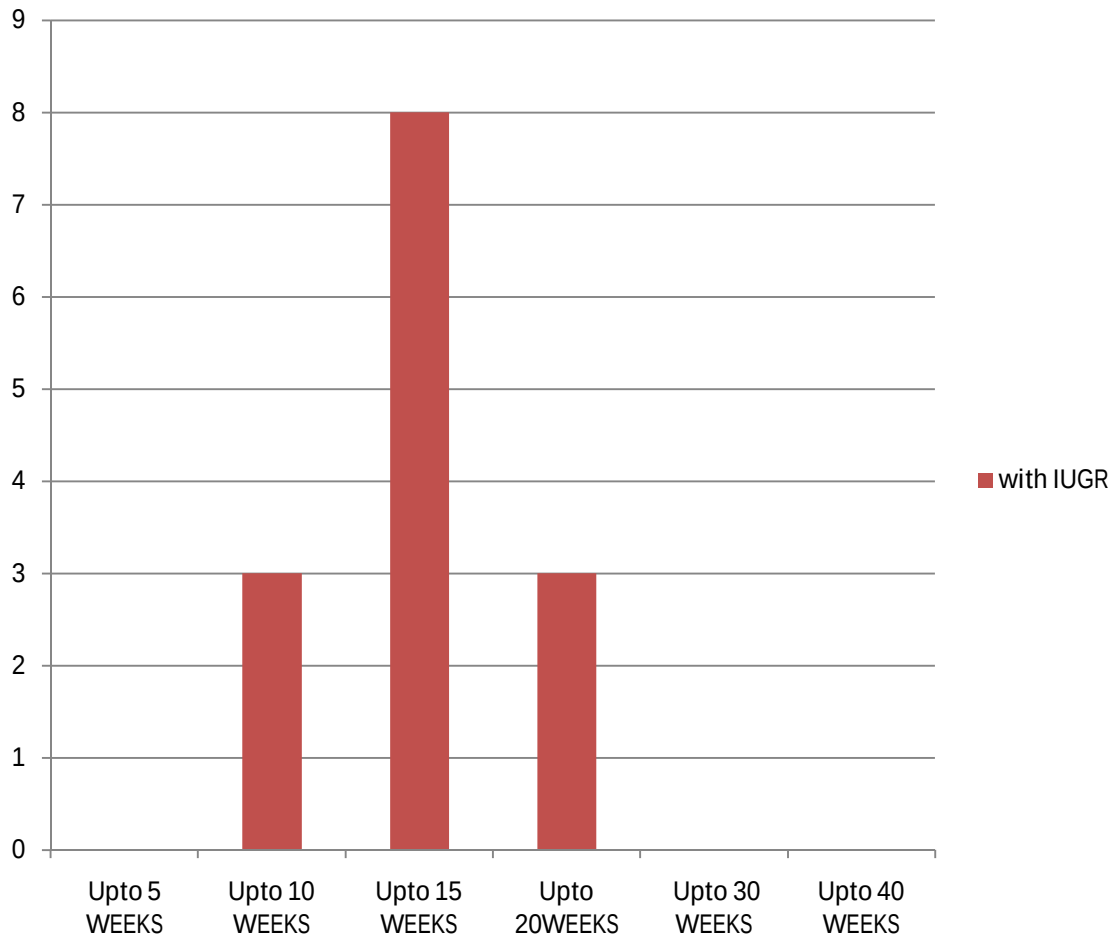
CORRELATION BETWEEN DURATION OF HYPEREMESIS GRAVIDARUM AND IUGR

Table 15

GESTATIONAL AGE	IUGR
Upto 5 WEEKS	0
Upto 10 WEEKS	3
Upto 15 WEEKS	8
Upto 20WEEKS	3
Upto 30 WEEKS	0
Upto 40 WEEKS	0
TOTAL	**14**

Of 80 patients with hyperemesis gravidarum 14 patients(17.5%) had intrauterine growth restriction. Out of these 14 patients 8 patients were hospitalized for hyperemesis gravidarum between 11-15 weeks gestation. .

CORRELATION BETWEEN DURATION OF HYPEREMESIS GRAVIDARUM AND INTRAUTERINE GROWTH RESTRICTION



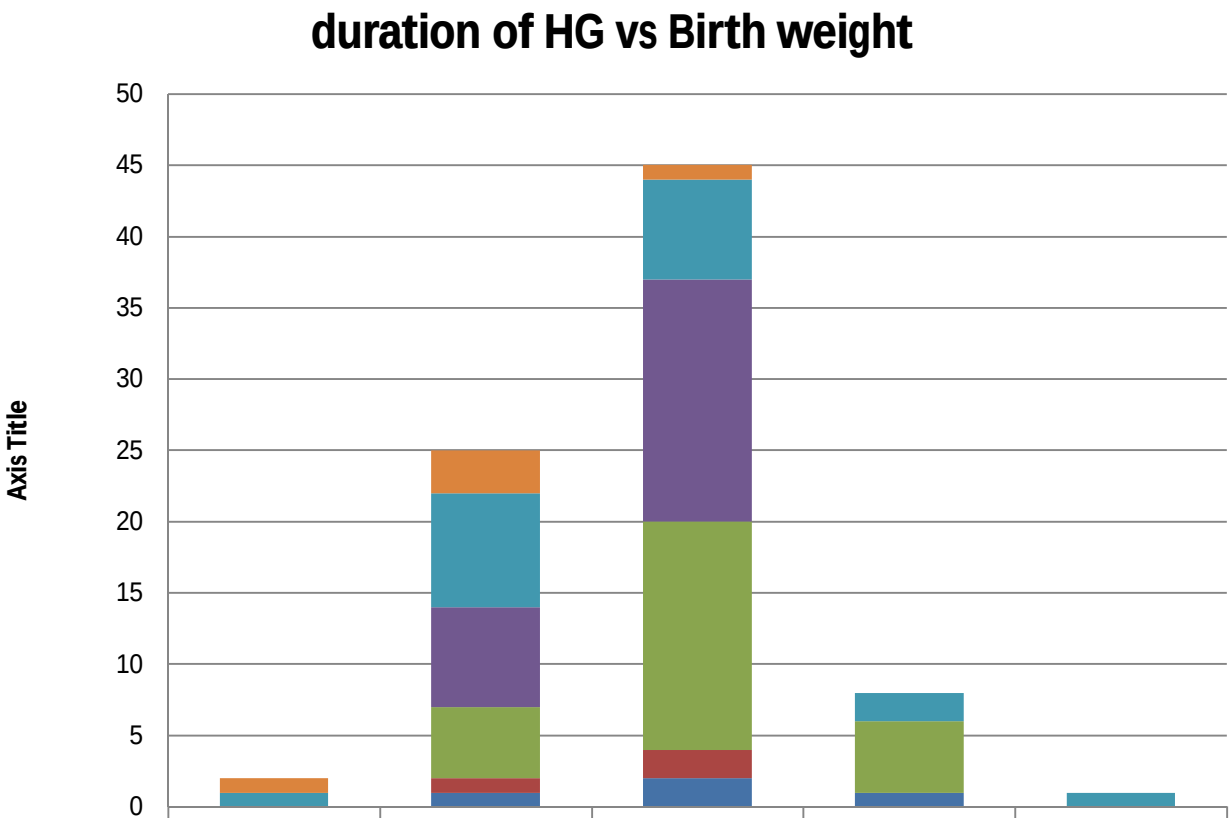
DURATION OF HYPEREMESIS Vs BIRTH WEIGHT

GESTATIONAL AGE	BIRTH WEIGHT					TOTAL
	1.5-2kgs	2.1-2.5kgs	2.6-3kgs	3.1-3.5kgs	3.6-4kgs	
Upto 5 WEEKS	0	0	0	1	0	1
Upto 10 WEEKS	1	5	7	8	3	24
Upto 15 WEEKS	2	17	16	7	1	43
Upto 20WEEKS	0	5	0	2	0	7
Upto 30 WEEKS	0	0	0	1	0	1
TOTAL	**3**	**27**	**23**	**19**	**4**	**76**

Out of 80 patients with hyperemesis gravidarum , 45 patients were hospitalized between 11- 15 weeks gestation . out of these 45 cases 2 pregnancies ended up in spontaneous abortion and 17 patients delivered low birth weight babies with birth weight weighing 2.1 -2.5 kgs.

Whereas only 7 patients were hospitalized between 16-20 weeks gestation of which 5 patients delivered babies with low birth weight.

From my study it implies that low birth was more common in patients in whom pregnancies were complicated by hyperemesis gravidarum at an advanced gestational age rather than in early gestation.



	Upto 5 WEEKS	Upto 10 WEEKS	Upto 15 WEEKS	Upto 20WEEKS	Upto 30 WEEKS
3.6-4 Kgs	1	3	1	0	0
3.1-3.5 Kgs	1	8	7	2	1
2.6-3 Kgs	0	7	17	0	0
2.1-2.5 Kgs	0	5	16	5	0
1.5-2 Kgs	0	1	2	0	0
abortion	0	1	2	1	0

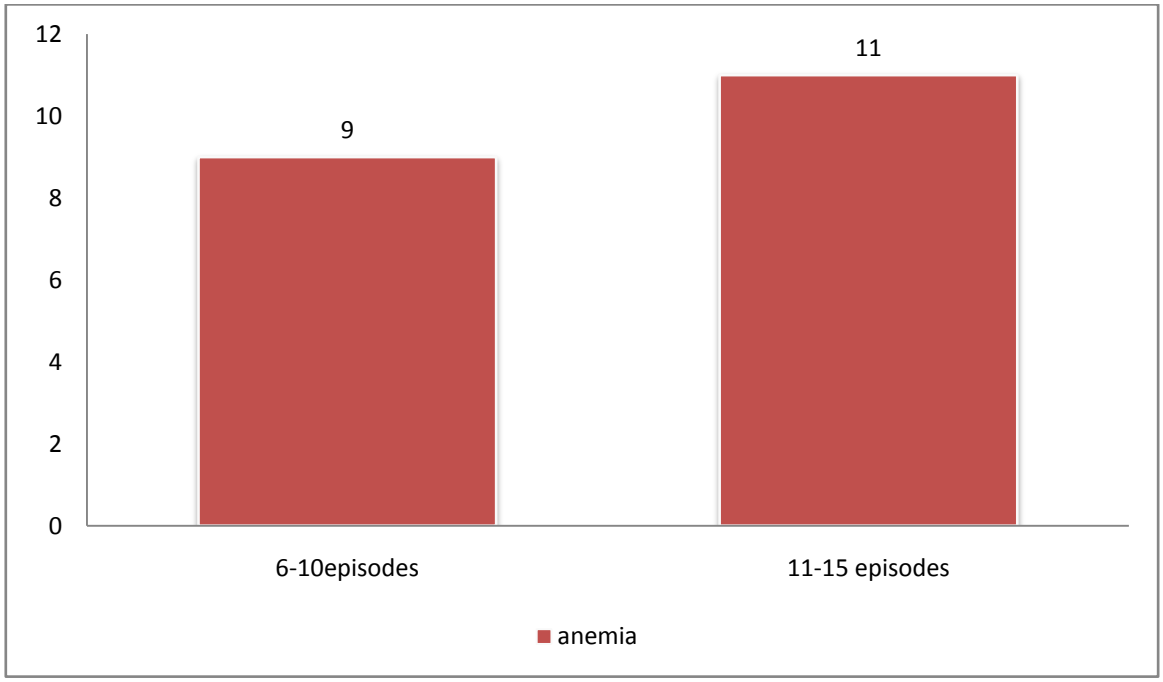
CORRELATION BETWEEN SEVERITY OF HYPEREMESIS GRVIDARUM AND ANEMIA

TABLE 17

NO OF EPISODES	ANEMIA	P value
6-10 episodes	9	0.302
11-15 episodes	11	
TOTAL	**20**	

Of 80 patients with HG , 20 patients developed anemia. Of these 20 patients ,anemia was seen in 11 patients who had 11-15 episodes of vomiting per day compared to 9 patients who had less than 10 episodes per day and was found to be statistically insignificant.

CHART 15



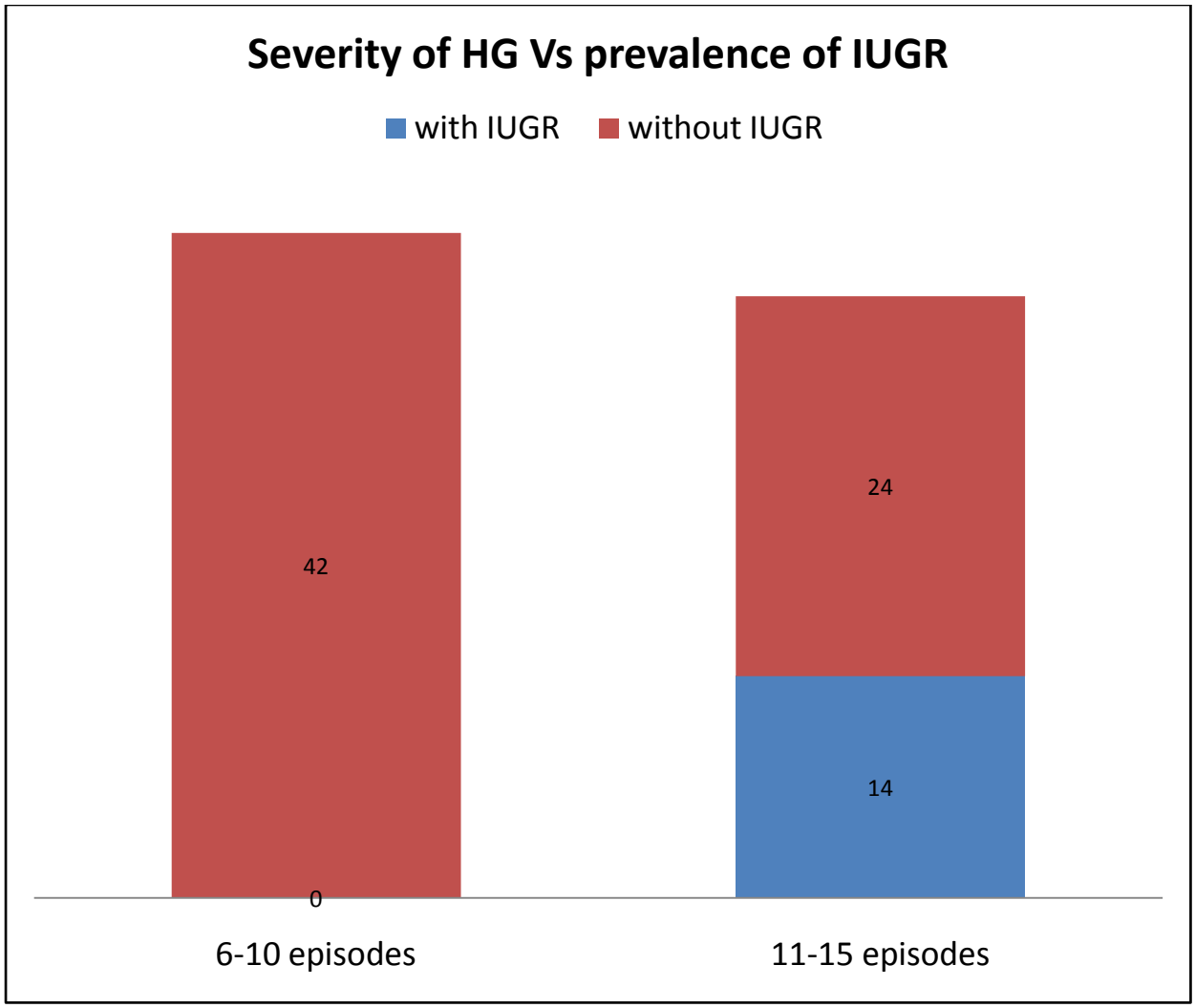
CORRELATION BETWEEN SEVERITY OF HYPEREMESIS GRVIDARUM AND INTRAUTERINE GROWTH RESTRICTION

TABLE 18

NO OF EPISODES	IUGR	P value
6-10 EPISODES	0	.000
11-15 EPISODES	14	

Of 80 patients with hyperemesis gravidarum 14 patients had intrauterine growth restriction all the 14 patients had 11-15 episodes of vomiting per day. Thus from my study it was found that IUGR was most commonly seen in patients with more than 10 episodes of vomiting per day and thus severe hyperemesis and its association with intrauterine fetal growth restriction was found to be statistically significant.

CHART 16



From my study intrauterine growth restriction itself was not significantly associated with hyperemesis gravidarum , but all the 14 patients with IUGR had severe hyperemesis which was proven to be statistically significant.

CORRELATION BETWEEN SEVERITY OF HYPEREMESIS GRAVIDARUM AND PREGNANCY WEIGHT GAIN

TABLE 19

NO OF EPISODES	WEIGHT GAIN				
	3-5 kgs	**6-8 kgs**	**9-11 kgs**	**12-14 kgs**	**19-21 kgs**
6-10 episodes	2	7	20	12	1
11-15 episodes	3	21	10	2	0
TOTAL	**5**	**28**	**30**	**14**	**1**

P value - .001 *

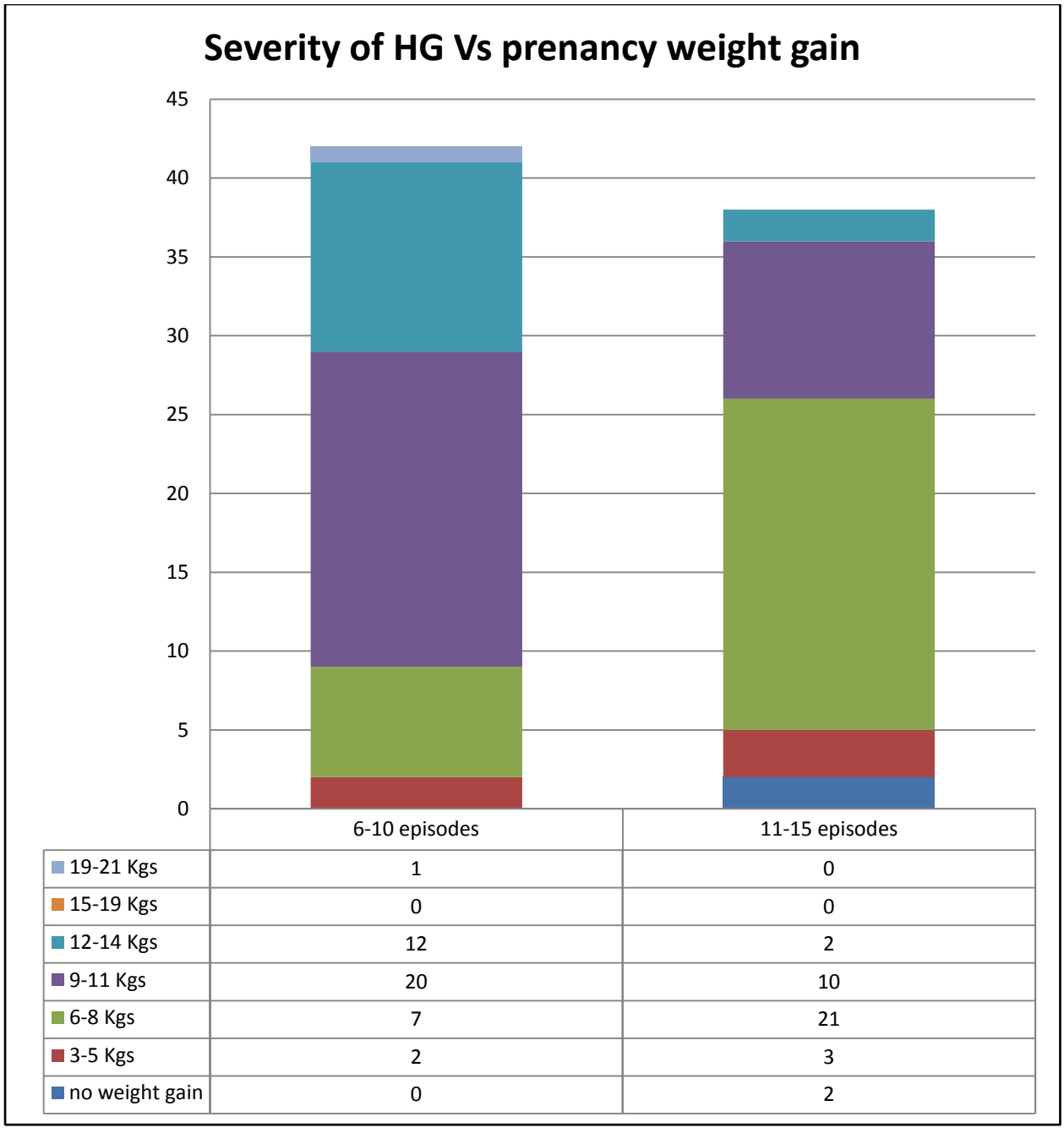
***statistically significant**

Patients with 6-10 episodes per day had a better weight gain of 9-11kgs when compared to those patients who had 11-15 episodes per day who had a weight gain of only 6-8 kgs.

There were totally 38 patients with 11-15 episodes of vomiting per day , of which 2 pregnancies resulted in abortion , 21 patients (55.5%) had a weight gain of only 6-8 kgs throughout the pregnancy.

CORRELATION BETWEEN SEVERITY OF HYPEREMESIS GRAVIDARUM AND PREGNANCY WEIGHT GAIN

CHART 17



	6-10 episodes	11-15 episodes
19-21 Kgs	1	0
15-19 Kgs	0	0
12-14 Kgs	12	2
9-11 Kgs	20	10
6-8 Kgs	7	21
3-5 Kgs	2	3
no weight gain	0	2

SEVERITY OF HYPEREMESIS GRAVIDARUM Vs BIRTH WEIGHT

TABLE 20

NO OF EPISODES	BIRTH WEIGHT					TOTAL
	1.5-2 kgs	2.1 -2.5 Kgs	2.6-3 kgs	3.1-3.5 kgs	3.6-4 kgs	
6-10 EPISODES	1	5	15	16	4	42
11-15 EPISODES	2	21	9	3	0	38

P value - < 0.01 *

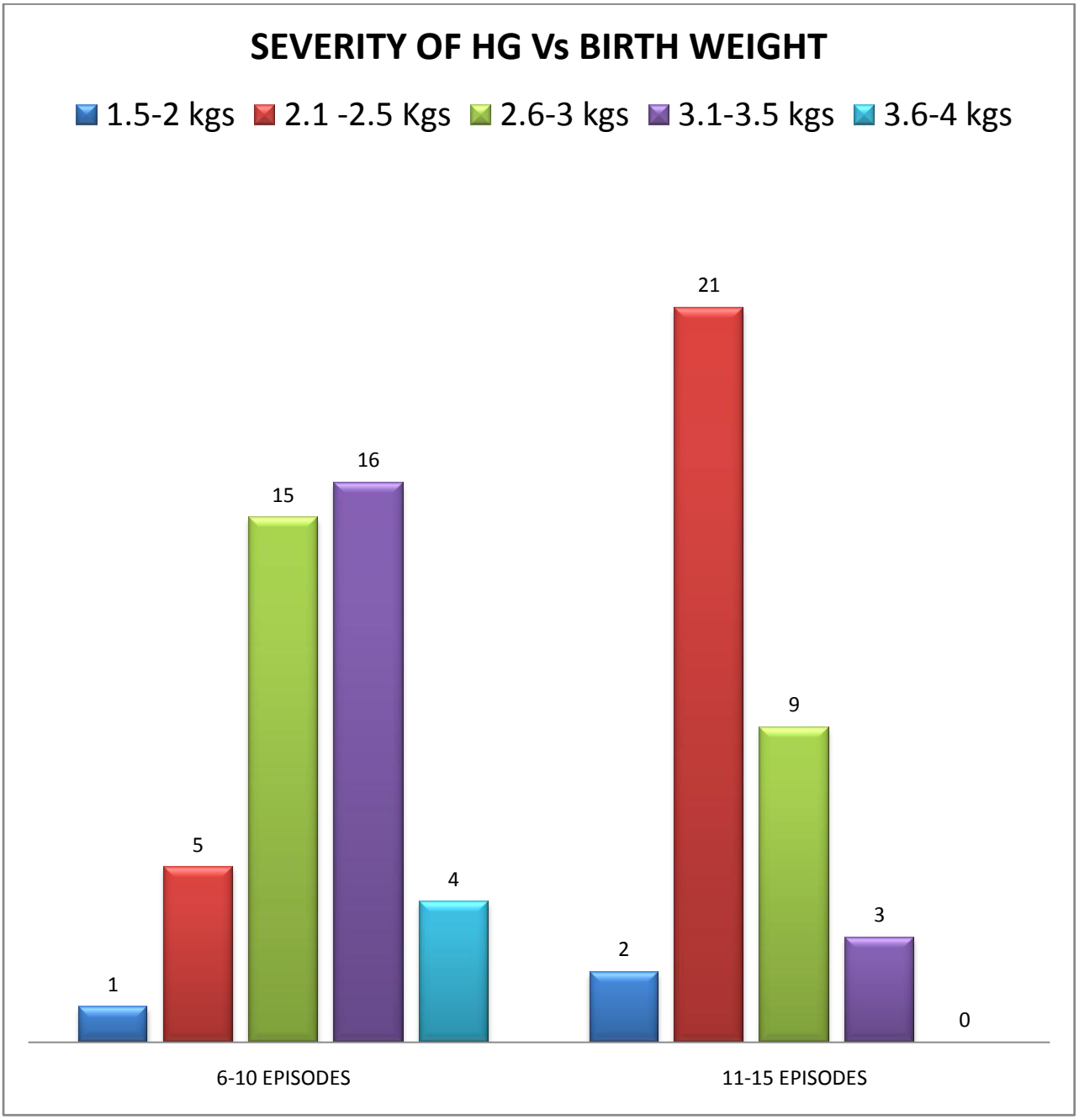
***Statistically significant**

Out of 38 patients with 11-15 episodes of vomiting per day 21 patients delivered babies with low birth weight ranging from 2.1-2.5 kgs which signifies that patients with severe vomiting are more prone for giving birth to low birth babies.

Whereas in patients with 6-10 episodes of vomiting most of the patients delivered babies with birth weight > 2.5 kgs.

SEVERITY OF HYPEREMESIS GRAVIDARUM Vs BIRTH WEIGHT

CHART 18



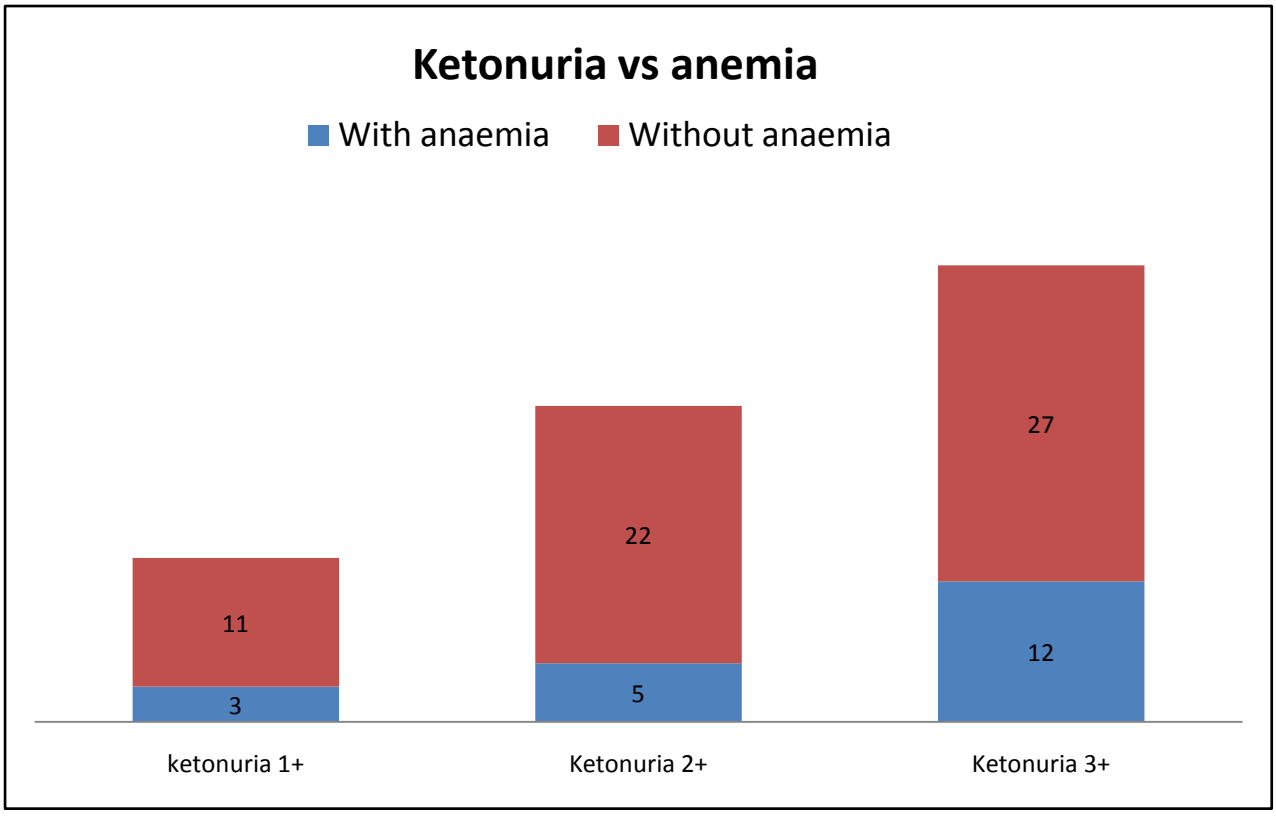
KETONURIA VS ANEMIA

TABLE 21

KETONURIA	ANEMIA	P value
1+	3	0.100
2+	5	
3+	12	
TOTAL	**20**	

The incidence of anemia was more in patients with ketonuria 3+. Out of 20 patients with anemia 12 had 3+ ketonuria but was not statistically significant.

CHART 19



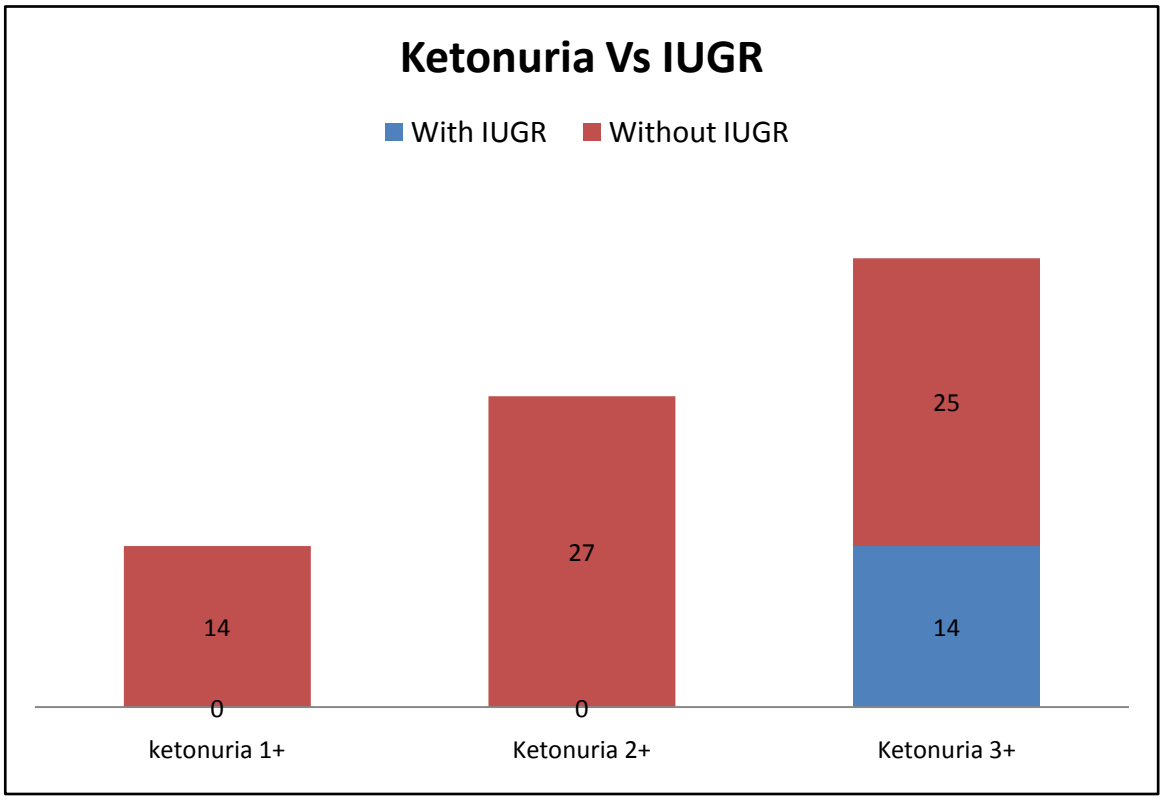
KETONURIA VS IUGR

TABLE 22

ketonuria	IUGR	P value
1+	0	0.000
2+	0	
3+	14	
total	**14**	

All 14 patients with intrauterine growth restriction had ketonuria 3+ and was statistically siagnificant. Thus signifying the importance of ketonuria in assessing the severity of hyperemesis gravidarum.

CHART 20



KETONURIA Vs BIRTH WEIGHT

TABLE 23

KETONURIA	BIRTH WEIGHT						TOTAL
	ABORTION	1.5-2 kgs	2.1-2 .5kgs	2.6-3 kgs	3.1-3.5 kgs	3.6-4 kgs	
1+	0	1	1	2	9	1	14
2+	1	0	2	12	9	3	27
3+	3	2	23	10	1	0	39

Totally 39 patients out of 80 had ketones 3 + in urine of which 28 patients had low birth weight of less than 2.5 kgs but the association of birth weigh and ketonuria was not significant in my study.

KETONURIA VERSUS BIRTH WEIGHT

CHART 21

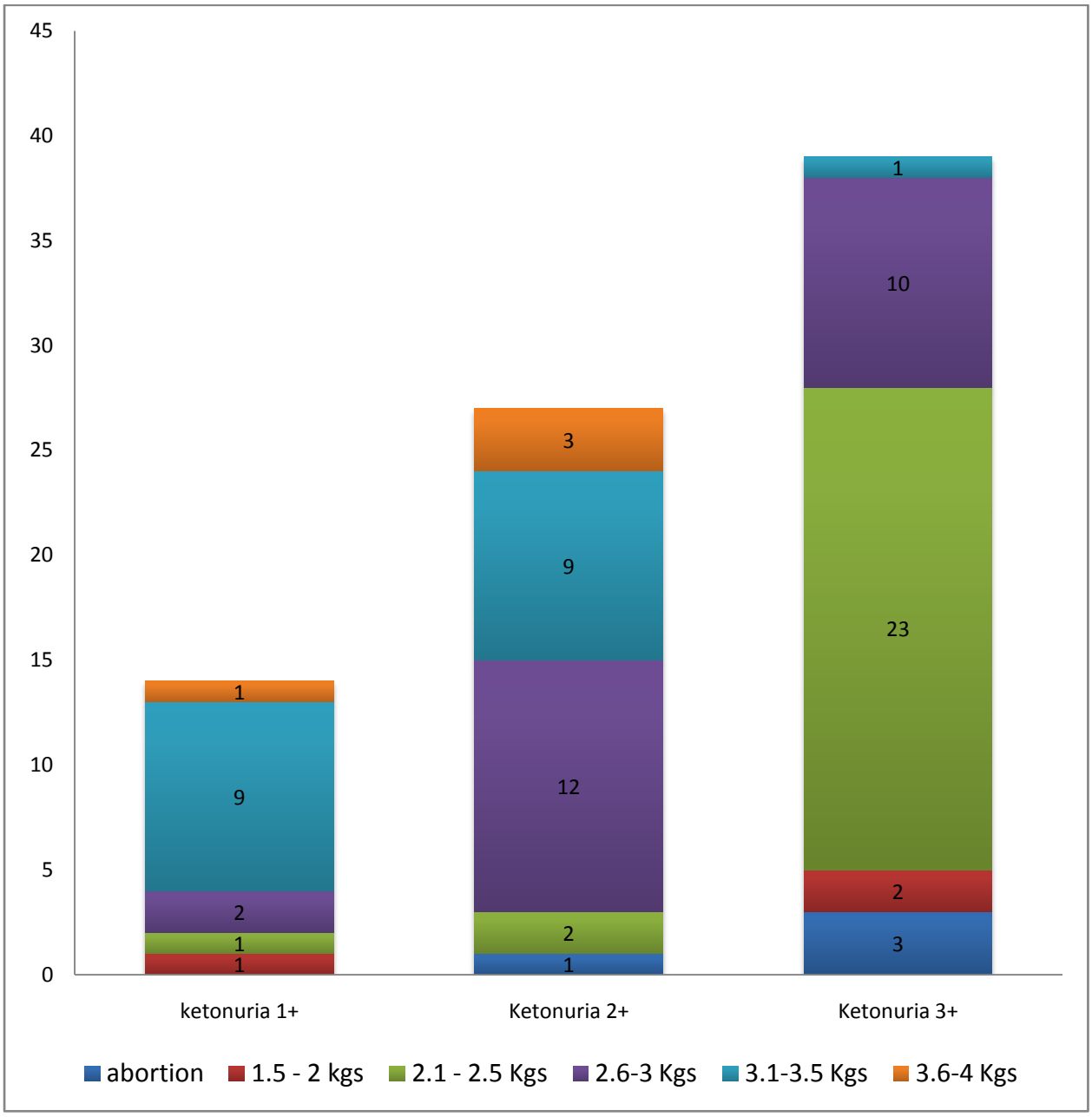
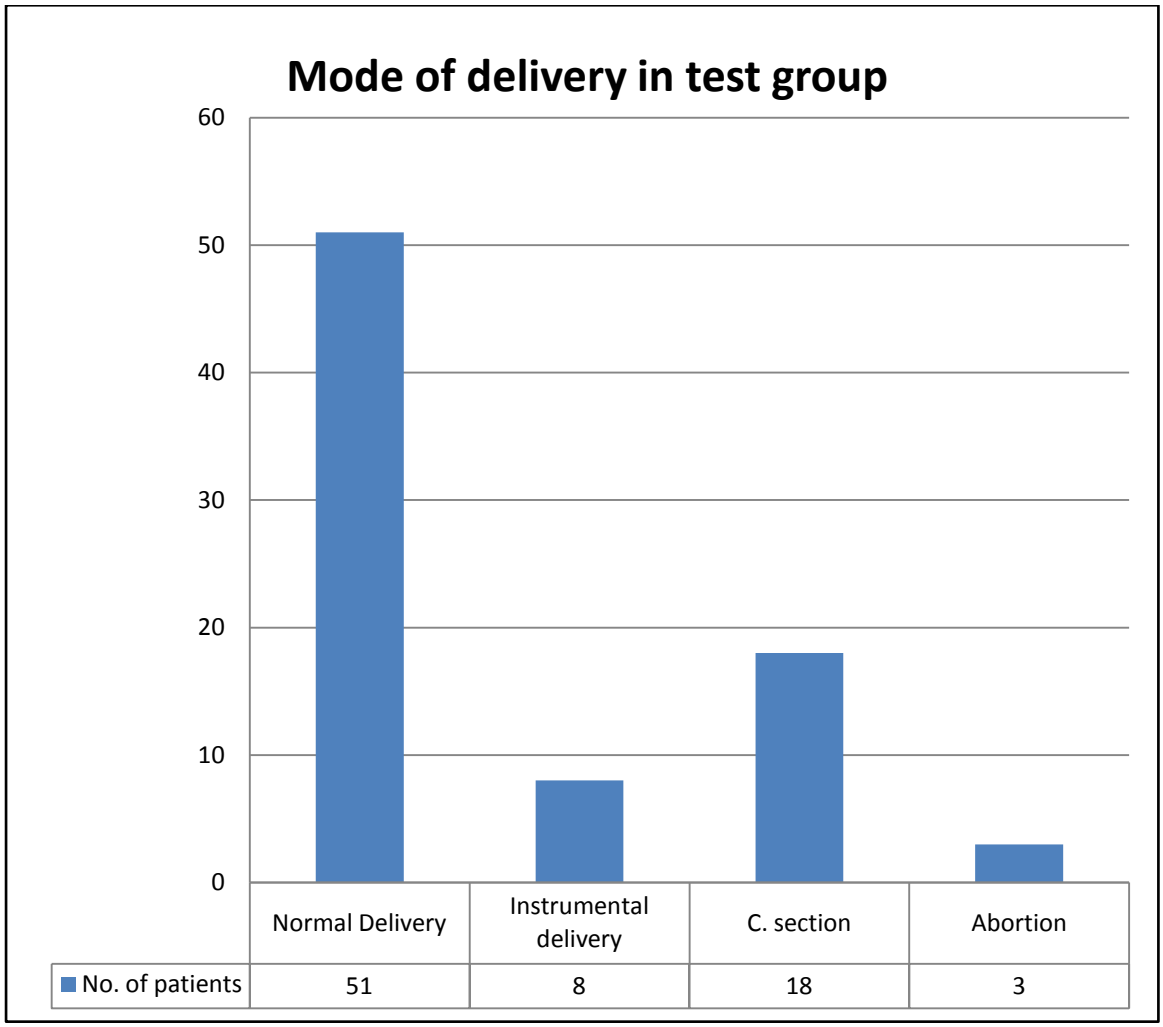


CHART 22

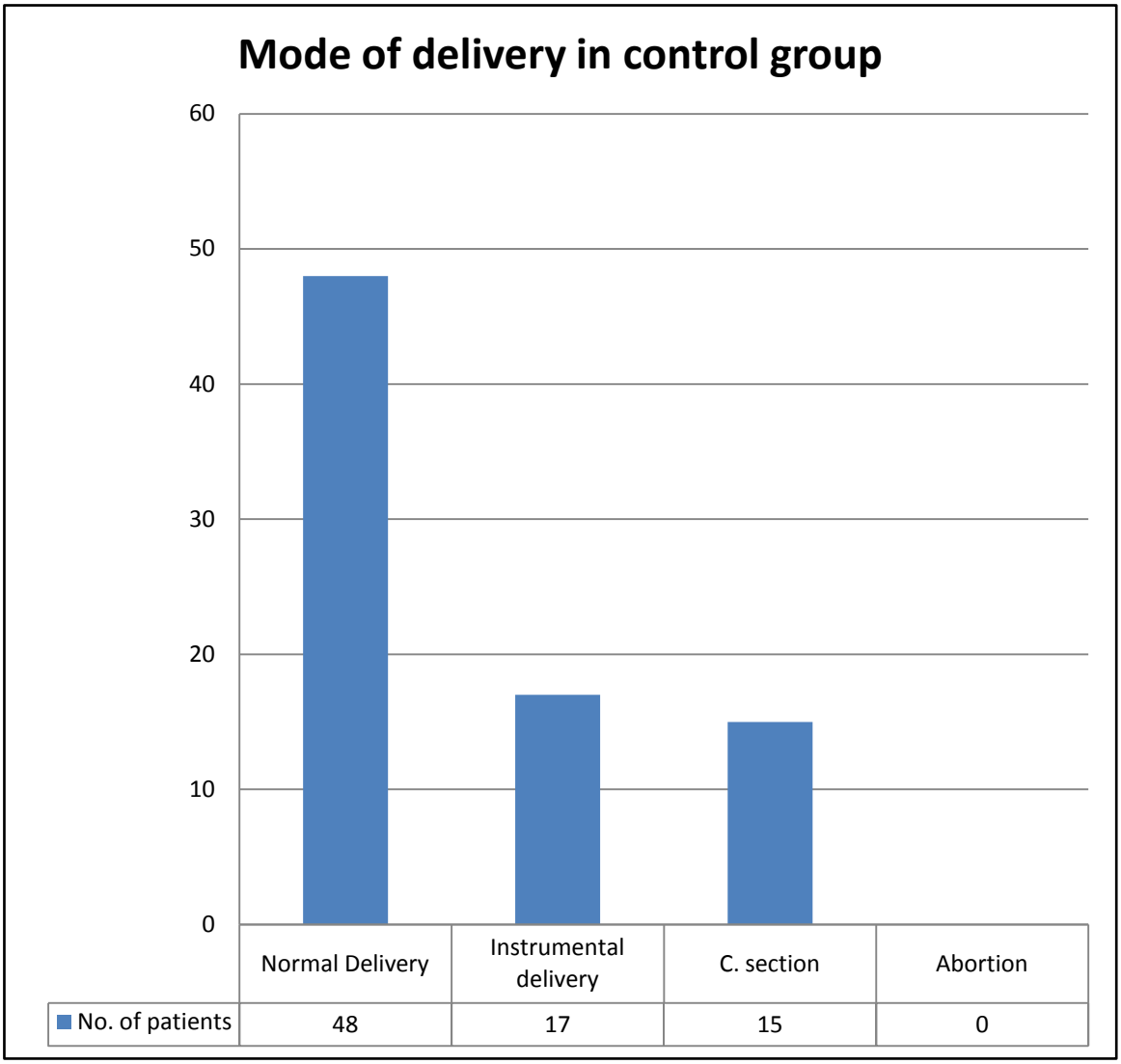


Percentage of Normal delivery - 63.8%

Percentage of instrumental delivery - 10 %

Percentage of C section - 22.5%

CHART 23



Percentage of normal delivery - 60%

Percentage of instrumental delivery – 21.3%

Percentage of C section – 18.75%

DISCUSSION

This prospective observational study presents a comprehensive profile on the maternal and fetal outcome in pregnancies complicated by hyperemesis gravidarum. As already discussed nausea and vomiting in pregnancy affects about 50-60% of pregnant mothers with variable severity and hyperemesis gravidarum affects about 0.5 – 2% of the pregnant mothers. As discussed previously the exact etiology of nausea and vomiting in pregnancy still a matter of debate. That has resulted in lot many proposals and theories in the literature. Few studies report that nausea and vomiting in pregnancy is associated with favourable pregnancy outcome[8-10]. On the other hand lot many studies have shown HG to be associated with adverse pregnancy outcomes like low birth weight , preterm birth, small-forgestational-age , prolonged stay in hospital for the new-born infant [1] and p erinatal death and [26-129].With this background we made an effort to study the impact of hyperemesis on maternal and fetal outcome, here at PSG hospitals Coimbatore. This is a prospective observational study conducted between June 2014 – 2015.

In our study hyperemesis was more common among younger age group women between 20 -25 yrs of age. About 51.25% of the patients with hyperemesis in our study group were in the age group of 20-25yrs. With advancing age the prevalence gradually decreases which is similar to the previous reports[14,132]. This could be due to increased prevalence of pregnancy in this age group especially in Indian population.In our study hyperemesisgravidarum was common among primi mothers when compared to multiparous women. The incidence of hyperemesis in nulliparous women was 65%

when compared to 35% in multiparous women. Similar parity preponderance was seen in many other studies including the large Norwegian cohort study[14,132]. This difference may be due to the fact that multiparous women, who had hyperemesis in the first pregnancy, cope up the symptoms much easier than a nulliparous mother who is very anxious about her pregnancy. It's always rare for a multiparous woman to have hyperemesis without having hyperemesis in her first pregnancy. The strongest risk factor for HG in pregnancy is having it in a previous pregnancy[119,120,133].

The prevalence of anemia among the patients who had hyperemesis was 25% which is significantly more when compared to 10 % in control group (p value < 0.1). Of 20 patients with hyperemesis gravidarum who developed anemia 13 patients were hospitalized for hyperemesis at 11-15 weeks. The incidence of anemia was more in the patients who had hyperemesis lasting till second trimester than those who had hyperemesis only upto 10 weeks of gestation but yet statistically insignificant. Similarly there is no significant relationship between number of episodes of vomiting or severity of ketonuria and prevalence of anaemia. Although the association between anaemia and hyperemesis is less frequently reported in the literature, because of the fact that anaemia in pregnancy by itself is multifactorial, the present observation in our study may be due to reluctance of accepting oral iron supplementation among mothers who have significant nausea and vomiting in pregnancy.

The prevalence of gestational diabetes mellitus is same in both study and control group (p value – 1.0) indicating that there is no significant association between hyperemesis and gestational diabetes which is similar to the observations found in a report from USA [134]. Similarly prevalence was the same for gestational

glucose intolerance (GGI) among the study and control group. The prevalence of GGI was 9% in both the groups. The prevalence of oligohydromnios was almost the same in both study and control group. It's really interesting that even in women who had significant dehydration due to hyperemesis , the incidence of oligodramnios is rare. This association may be due the fact that hyperemesis is very rare in third trimester but most of the oligohydromnios manifest significantly in the third trimester.

There were no significant difference in the prevalence of intra uterine growth restriction (IUGR) /Small for gestational age (SGA) in our study among the two groups. 14/80 women who had hyperemesis had SGA babies but even in the control group who had no hyperemesis, 12/80 mothers had SGA babies. Similarly patient with prolonged duration of hyperemesis also had no significant correlation with IUGR. This observation is similar to few reports found in literature [128,131]. But majority of the studies have found a positive association between hyperemesis and delivered babies who were small for gestational age [126-129]. This observation may be due to the fact that, this study was conducted in a tertiary care private sector hospital where majority of the patients are economically strong as well as educated and thus have good compliance to the advised treatment and supportive care. Still a prospective study with a larger group involving many centers is worth performing to assess the actual impact of hyperemesis on fetal outcome.

The pregnancy weight gain among the mothers who had hyperemesis was less when compared to the control group which is similar to the study done among Norwegian mothers [132]. But this observation was not found significant. 5/80 mothers who had hyperemesis had preterm delivery when compared to 2/80 mothers who had

no hyperemesis. 3 patients in the study group had spontaneous first trimester abortion. This association was also not found significant. This observation was similar to the study conducted in Norwegian mother [132]. But lot many other studies [126-129]have found a significant positive association between hyperemesis and preterm deliveries. 37.5% of mothers among the study group had low birth weight babies (< 2.5 Kgs) compared to 26.25 % in the control group. This association is yet statistically insignificant which is similar to observations noted in few studies [132,135-137] but few studies have reported a positive correlation between hyperemesis and low birth weight babies[126-129].

There is no significant correlation between duration of hyperemesis and prevalence of gestational hypertension or gestational diabetes mellitus although the numbers are little high in patients who had hyperemesis lasting till second trimester than who had symptoms restricted to first trimester. In our study there were no significant correlation between duration of hyperemesis symptoms and birth weight although number of low birth weight babies are more in patients who had symptoms prolonging till second trimester, similar to the observations noted in a study from Norway [132].

Patients who had increased frequency of vomiting episodes tend to have IUGR babies in our study. That is those patients who had more that 10 episodes of vomiting in a day and those who had severe ketonuria significantly had greater propotion of IUGR/ SGA babies (P value -0) which is similar to the observations noted in few studies [126-129.]Similarly birthweight of the baby was correlating significantly with the number episodes of vomiting and severeity of ketonuria . That is mothers who had increased frequency of vomiting and those who had severe ketonuiria were found to

have low birthweight babies (p value – 0). We found a significant relationship between the pregnancy weight gain and number of episodes of vomiting. That is mothers who had greater frequencies of vomiting episodes tend to gain less weight during the entire period of pregnancy ($p<0.01$).

CONCLUSION

In the present study,

- Hyperemesis is noted predominantly in younger primi mothers.
- Anaemia is significantly higher among the mothers who had hyperemesis gravidarum.
- Among the study group, mothers who had more than 10 episodes of vomiting in a day and those who had severe ketonuria significantly had greater proportion of IUGR/ SGA babies.
- Similarly those mothers who had more than 10 episodes of vomiting with significant ketonuria had low birth weight babies.
- Mothers who had increased episodes of vomiting had decreased overall pregnancy weight gain.
- There is no significant association between hyperemesis gravidarum and preterm births.
- Similarly hyperemesis is not associated with IUGR or small for gestational age and low birth weight babies.
- There was no significant difference in the apgar score at 1^{st} and 5^{th} minute in both the study group and control group.

Thus etiology of nausea and vomiting in pregnancy is multi factorial, involving psychological changes, evolutionary adaptation, hormonal stimuli, and may be H.

pylori infection also. None is proved yet. Management of these symptoms depends on severity of symptoms, impact of symptoms on a woman's quality of life, and safety of the fetus. In our study just mere presence of hyperemesis requiring admissions doesn't have any adverse pregnancy outcome. But if the symptoms are severe with significant ketonuria, adverse outcomes like IUGR, low birth weight, poor maternal weight gain are expected. Still a study with a larger group involving multiple centres is needed to further analyze the actual impact.

STATISTICAL METHODS

Descriptive and inferential statistical analysis is carried out in this study. Results on continuous measurements are represented as Mean +/- SD (min-max) and results on categorical measurements are represented as numbers (%). Significance between both the groups are assessed at 5% significance level. The following data assumptions are made: 1. Dependant variables are normally distributed among both the groups. 2. Samples drawn from the population are random; cases of the samples are independent.

Student t test (independent, two tailed) was used to find the significance of the parameters in the study on a continuous scale between both the groups (inter group analysis) on metric parameters. Fisher exact test/ chi square test were used to find the significance among the parameters in this study on categorical scale between both the groups.

Significant figures:

+ Suggestive significance (P value: $0.05<P<0.10$)

*Moderately significant (P value: $0.01<P<0.05$)

**Strongly significant (P value: $P<0.01$)

Statistical software: The statistical software namely SPSS were used for the data analysis and Microsoft excel was used to generate tables, graphs and charts.

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PROFORMA

Pt name:

Age :

IP no :

Obstetric score:

GA at which hospitalised for HG:

- No of episodes/day
- Dehydration : yes no
- Urine ketones : positive negative

Antenatal complications if any:

Weight gain during pregnancy:

GA at time of delivery :

Mode of delivery:

Birth weight :

Apgar score : 1min 5 min

PSG Institute of Medical Science and Research, Coimbatore
Institutional Human Ethics Committee
INFORMED CONSENT FORMAT FOR RESEARCH PROJECTS

(strike off items that are not applicable)

I / We (write name of the investigator(s) here), _S.SUJITHRA am carrying out a study on the topic: HYPEREMESIS GRAVIDARUM AND PREGNANCY OUTCOME

as part of my / our research project being carried out under the aegis of the Department of: OBSTETRICS AND GYNAECOLOGY

My / our research guide is: **DR.CHITRA . T.V MD,DGO,DNB**

The justification for this study is:
Hyperemesis gravidarum has been reported to be associated with increased risk of low birth weight , small for gestational age and preterm birth. All these factors underline the necessity to study its potential effect on pregnancy outcomes.
The objectives of this study are:

To study the maternal and fetal outcome in pregnancies complicated by hyperemesis gravidarum as compared to those without hyperemesis:

Sample size: cases 80 ; control 80

Location: PSGIMS&R.

We request you to kindly cooperate with us in this study. We propose collect background information and other relevant details related to this study. We will be carrying out:

Initial interview (specify approximate duration):10 minutes.

Data collected will be stored for a period of fifteen years. We will / will not use the data as part of another study.

Specify **purpose**, discomfort likely to be felt and side effects, if any: _NA

Final interview (specify approximate duration):10mts.

Benefits from this study: Awareness about hyperemesis gravidarum among patients

Risks involved by participating in this study: NIL

How the **results** will be used: dissertation

If you are uncomfortable in answering any of our questions during the course of the interview / biological sample collection, **you have the right to withdraw from the interview / study at anytime.** You have the freedom to withdraw from the study at any point of time. Kindly be assured

that your refusal to participate or withdrawal at any stage, if you so decide, will not result in any form of compromise or discrimination in the services offered nor would it attract any penalty. You will continue to have access to the regular services offered to a patient. You will **NOT** be paid any remuneration for the time you spend with us for this interview / study. The information provided by you will be kept in strict confidence. Under no circumstances shall we reveal the identity of the respondent or their families to anyone. The information that we collect shall be used for approved research purposes only. You will be informed about any significant new findings - including adverse events, if any, – whether directly related to you or to other participants of this study, developed during the course of this research which may relate to your willingness to continue participation.

Consent: The above information regarding the study, has been read by me/ read to me, and has been explained to me by the investigator/s. Having understood the same, I hereby give my consent to them to interview me. I am affixing my signature / left thumb impression to indicate my consent and willingness to participate in this study (i.e., willingly abide by the project requirements).

Signature / Left thumb impression of the Study Volunteer / Legal Representative:

Signature of the Interviewer with date: Witness:

Contact number of PI: 9843334041

Contact number of Ethics Committee Office: 0422 2570170 Extn.: 5818

Sl. No	NAME	AGE	IP NO	OBS SCORE	GA OF HYPEREMESIS	NO OF EPISODES	DEHYDRATION	KETONURIA	ANEMIA	GDM	GGI	GESTATIONAL HT	ABORTION	OLIGOHYDRAMNIOS	IUGR	AN COMPLICATIONS	WEIGHT GAIN	GA AT DELIVERY	NVD	INSTRUMENTAL DELIVERY	LSCS	BIRTH WGT	APGAR 1 MIN	APGAR 5 MIN
1	krishnaven	1	I14034961	1	3	2	1	2	0	1	0	0	0	1	0	1	1	3	1	0	0	2	3	3
2	sowmya	1	i14036811	1	3	2	0	2	0	0	1	0	0	0	0	1	1	3	1	0	0	3	3	3
3	shanthi	3	I15001643	2	2	2	0	1	0	0	0	0	0	0	0	0	3	3	0	0	1	1	3	3
4	prabhavath	4	I15002190	1	2	2	1	3	0	0	0	0	0	0	0	0	3	3	0	0	1	3	3	3
5	gowri	2	I15002773	2	2	2	1	2	0	0	0	0	0	0	0	0	3	3	1	0	0	3	3	3
6	vijitha	1	I15007133	1	3	2	1	3	0	0	0	0	0	0	0	0	4	3	0	1	0	3	3	3
7	poornima	1	I15007503	1	3	3	1	3	0	0	1	0	0	0	0	1	4	3	1	0	0	4	3	3
8	sofia madh	1	I15007729	1	2	2	0	2	1	0	0	0	0	0	0	1	6	3	0	0	1	4	3	3
9	kavitha	2	I15009077	2	6	2	1	1	1	0	0	0	0	1	0	1	2	3	0	1	0	4	3	3
10	manoshree	1	I15009502	1	2	3	1	3	0	0	0	0	0	0	1	1	2	3	1	0	0	2	3	3
11	ruby	3	I15009744	2	2	2	0	1	1	0	0	0	0	0	0	1	2	3	1	0	0	3	3	3
12	jeeva rathi	1	I15009487	1	3	2	1	2	0	0	0	0	0	0	0	0	4	3	1	0	0	3	3	3
13	malathi	1	I15009742	1	2	2	1	2	0	0	0	0	0	0	0	0	3	3	0	0	1	4	2	3
14	poongodi	3	I14040053	1	4	2	0	1	0	0	0	0	0	0	0	0	4	3	0	0	1	2	3	3
15	shine josep	4	I14036953	2	2	2	1	2	0	0	0	0	0	0	0	0	3	3	1	0	0	4	3	3
16	poornima c	2	I15002531	2	3	2	1	1	0	0	0	0	0	0	0	0	2	3	1	0	0	4	3	3
17	fathima	1	I15004616	1	3	2	1	3	0	0	0	0	0	0	0	0	2	2	1	0	0	2	3	3
18	susheela	3	I15008774	2	3	2	1	2	0	1	0	0	0	0	0	1	3	3	1	0	0	0	3	3
19	Kavitha	1	I15011942	1	3	3	1	3	1	0	0	0	0	0	0	1	2	3	0	0	1	3	3	3
20	Pradeepa	1	I14034825	1	3	3	1	3	0	0	0	0	1	0	0	1	0	0	0	0	0	0	0	0
21	gayathri	1	I15003383	1	3	2	1	2	0	0	0	1	0	0	0	1	2	3	1	0	0	4	2	3
22	nagaothi	1	I15004041	1	2	2	1	2	0	0	0	0	0	0	0	0	3	3	0	0	1	5	3	3
23	Kavitha	2	I15005311	1	2	2	1	2	0	0	0	0	0	0	0	0	3	3	0	1	0	4	3	3
24	divya	3	I15007457	2	3	3	0	3	0	0	0	0	0	0	1	1	2	3	1	0	0	2	3	3
25	safaa basha	1	I15008086	1	4	3	1	3	0	0	0	0	0	0	1	1	2	3	0	1	0	2	3	3
26	suganya	2	I15079646	1	2	2	1	3	1	0	0	0	0	0	0	1	2	2	1	0	0	2	2	3
27	bhuvanesh	1	I15023882	1	2	2	0	2	0	0	0	0	0	0	0	0	3	3	1	0	0	3	3	3
28	santhanam	3	I14029206	2	2	3	1	3	1	0	0	0	0	0	1	1	1	3	1	0	0	2	3	3
29	Priya g	2	I15036964	1	4	3	1	3	0	0	0	0	0	0	0	0	3	2	1	0	0	2	3	3
30	revathi	1	I14010878	1	3	2	0	2	0	0	0	0	0	0	0	0	4	3	1	0	0	3	3	3
31	krithika	1	I14011430	1	3	2	0	2	0	0	0	0	0	0	0	0	3	3	1	0	0	3	3	3
32	sindhuja K	1	I13032737	1	4	3	0	2	1	0	1	0	0	0	0	1	2	3	1	0	0	4	3	3
33	deepika I	2	I14017145	2	3	3	1	3	1	0	0	0	0	0	0	1	3	3	1	0	0	3	3	3
34	umarani	2	I14016486	2	4	2	1	2	0	0	0	0	0	0	0	0	3	3	1	0	0	4	3	3
35	sathyadevi	2	I14015075	2	2	2	0	2	0	1	0	0	0	0	0	1	4	3	0	0	1	5	3	3
36	anandhi	1	I14014558	1	3	2	0	2	1	0	0	0	0	0	0	1	3	3	0	0	1	4	3	3
37	sowmiya I	1	I14021047	1	3	3	1	3	1	1	0	0	0	0	0	1	2	3	1	0	0	2	3	3
38	nithya	1	I14014138	1	3	3	1	3	0	0	0	0	0	0	0	0	2	2	0	1	0	1	3	3
39	radhika G	2	I14016336	2	3	2	0	1	0	0	0	0	0	0	0	0	3	3	0	1	0	4	3	3
40	kavitha	1	i15011942	1	2	2	1	3	1	0	0	0	0	0	0	1	3	3	0	0	1	3	3	3
41	priya	1	I15015838	1	2	3	1	2	0	0	0	0	0	0	0	0	4	3	0	0	1	3	3	3
42	dhanabhag	2	I15022115	2	2	2	0	2	0	0	1	0	0	0	0	1	3	3	0	0	1	4	3	3
43	punitha	1	I15013086	1	2	2	0	1	0	0	0	0	0	0	0	0	3	3	1	0	0	4	3	3
44	gomathi	2	I14032710	1	3	3	1	3	0	1	0	0	0	0	0	1	3	3	1	0	0	3	3	3
45	kalpana	2	I14026247	1	4	3	1	3	0	0	0	0	0	1	1	1	2	3	1	0	0	2	3	3
46	menaka	2	I14033769	2	2	2	0	1	0	0	0	0	0	0	0	0	4	3	1	0	0	4	3	3
47	neelavani	1	I14030540	1	3	3	1	2	0	0	0	0	0	0	0	0	3	3	1	0	0	3	2	3
48	ramya	2	I15009491	1	3	3	1	3	0	0	0	0	0	0	1	1	2	3	1	0	0	2	3	3
49	mahalaksh	3	I15009907	2	3	3	1	2	0	0	0	0	0	0	0	0	3	3	1	0	0	3	3	3
50	karthika	3	I15009989	2	2	3	1	3	0	0	0	0	0	0	0	0	3	3	1	0	0	2	3	3
51	nandhitha	1	I15009762	1	3	3	1	3	1	0	0	0	0	1	0	1	2	3	1	0	0	2	3	3
52	sowmya	2	I15010590	1	3	3	1	2	0	0	0	0	0	0	0	0	3	3	1	0	0	2	3	3
53	nazira	1	I15010796	1	3	3	1	3	0	1	0	1	0	0	1	1	1	3	1	0	0	1	3	3
54	dharanya	1	I15010863	1	3	3	1	3	0	0	0	0	0	0	1	1	2	3	1	0	0	2	3	3

Sl. No	NAME	AGE	IP NO	OBS SCORE	GA OF HYPEREMESIS	NO OF EPISODES	DEHYDRATION	KETONURIA	ANEMIA	GDM	GGI	GESTATIONAL HT	ABORTION	OLIGOHYDRAMNIOS	IUGR	AN COMPLICATIONS	WEIGHT GAIN	GA AT DELIVERY	NVD	INSTRUMENTAL DELIVERY	LSCS	BIRTH WGT	APGAR 1 MIN	APGAR 5 MIN
55	ramya	3	I15011020	2	2	3	1	3	0	0	0	1	0	0	1	1	2	3	1	0	0	2	2	3
56	usha	1	i15011646	1	1	2	0	1	0	0	0	0	0	0	0	0	4	3	0	1	0	4	2	3
57	rubini	2	I15012134	1	3	3	1	3	0	0	1	0	0	0	1	1	2	3	0	0	1	2	2	3
58	pavithra	1	I15011891	1	3	3	1	3	1	0	0	0	0	0	1	1	2	3	1	0	0	2	3	3
59	Uthyadevi	1	I14021705	1	3	2	0	1	0	0	0	0	0	0	0	0	4	3	1	0	0	4	3	3
60	anua	2	I14022011	2	3	2	0	2	0	0	0	0	0	0	0	0	3	3	1	0	0	3	3	3
61	hellen	1	I14022345	1	3	2	1	3	0	0	1	0	0	0	0	1	3	3	0	0	1	3	3	3
62	sadana	3	I14022764	2	3	2	1	2	1	0	0	0	0	0	0	1	3	3	1	0	0	5	3	3
63	nagalakshn	1	I14022706	1	2	2	0	1	0	0	0	0	0	0	0	0	4	3	1	0	0	4	3	3
64	kavitha	3	I14021658	2	3	3	1	3	0	1	0	0	0	0	0	1	2	3	1	0	0	2	3	3
65	parimal	2	I14022257	1	3	3	0	1	1	0	1	0	0	0	0	1	3	3	1	0	0	4	3	3
66	radhika	1	I14023930	1	3	3	1	3	1	0	0	0	0	1	1	1	2	3	1	0	0	3	2	3
67	priskilal	2	I14024985	2	3	3	1	3	0	1	0	0	0	0	0	1	3	2	1	0	0	2	3	3
68	sabina	2	I14023243	2	3	2	0	2	1	1	0	0	0	0	0	1	3	3	1	0	0	3	3	3
69	akalya	2	I14025130	2	3	3	1	3	0	0	0	0	0	0	1	1	2	3	1	0	0	2	2	3
70	padma	1	I14025730	1	3	3	1	3	0	0	0	0	0	0	0	0	3	3	0	1	0	3	3	3
71	triveni	2	I14025758	2	2	2	0	1	0	0	0	0	0	0	0	1	4	3	0	0	1	3	3	3
72	deepa	2	I14026944	2	3	2	1	3	1	0	0	0	0	0	0	1	2	3	0	0	1	2	2	3
73	Pavithra	1	I14028165	1	3	3	1	3	1	0	0	0	0	0	0	1	2	3	1	0	0	2	2	3
74	Gayathri	2	I14030160	2	3	2	0	2	0	0	0	0	0	0	0	0	4	3	1	0	0	3	3	3
75	seena	1	I14048019	1	4	3	1	3	0	0	0	0	1	0	0	1	1	0	0	0	0	0	0	0
76	kavitha	1	I15011942	1	3	3	1	3	1	0	0	0	0	0	0	1	2	3	0	0	1	3	3	3
77	sasikala	1	I15012284	1	3	3	1	3	0	0	0	0	0	0	0	0	2	3	0	0	1	2	2	3
78	Pavithra	1	I15012605	1	4	3	1	3	0	0	0	0	0	1	1	1	2	3	1	0	0	2	3	3
79	sudha	2	I14009029	2	2	2	0	1	0	0	0	0	0	0	0	0	4	3	1	0	0	5	3	3
80	Deepa	1	I14009487	1	2	3	1	3	0	0	0	0	1	0	0	1	0	0	0	0	0	0	0	0
81	Safiya	1	I14015309	1	0	0	0	0	0	0	0	0	0	0	0	0	4	3	0	0	1	3	3	3
82	reshma	3	I14015375	2	0	0	0	0	1	0	0	0	0	0	0	1	3	3	1	0	0	3	3	3
83	yazhini	1	I14015327	1	0	0	0	0	1	0	0	0	0	0	0	1	4	3	0	1	0	4	3	3
84	kanchana	1	I14015609	1	0	0	0	0	0	0	1	0	0	0	0	1	3	3	1	0	0	2	2	2
85	ruckmani	1	I14016533	1	0	0	0	0	0	1	0	0	0	1	1	1	3	2	1	0	0	2	3	3
86	suguna	3	I14017156	2	0	0	0	0	0	0	0	0	0	0	0	0	4	3	1	0	0	4	3	3
87	krithika	2	I14017075	2	0	0	0	0	0	0	0	0	0	0	0	0	3	3	1	0	0	4	3	3
88	Syeed ali	1	I14017580	1	0	0	0	0	0	0	0	0	0	0	1	1	3	3	1	0	0	2	3	3
89	shrithi	1	I14017686	1	0	0	0	0	0	0	0	0	0	0	0	0	4	3	1	0	0	4	3	3
90	jothi	2	I14017952	1	0	0	0	0	0	0	0	0	0	0	0	0	3	3	1	0	0	3	2	3
91	sangeetha	1	i14018522	1	0	0	0	0	0	0	0	0	0	0	0	0	3	3	0	1	0	3	3	3
92	Amutha	3	I14018703	2	0	0	0	0	0	0	0	0	0	0	0	0	4	3	0	0	1	3	2	3
93	Divya	1	I14018716	1	0	0	0	0	0	0	0	0	0	0	1	1	2	3	0	0	1	2	2	3
94	Gomathi	1	I14019003	1	0	0	0	0	0	0	0	0	0	0	0	0	4	3	0	1	0	3	3	3
95	sudha	2	I14019131	2	0	0	0	0	0	0	0	0	0	0	0	0	3	2	1	0	0	1	3	3
96	sathya	1	I14023420	1	0	0	0	0	0	0	0	0	0	0	0	0	4	3	0	0	1	5	3	3
97	archana	2	I14019321	2	0	0	0	0	0	0	0	0	0	0	0	0	3	3	1	0	0	3	3	3
98	Gowri	1	I14019540	1	0	0	0	0	0	0	0	0	0	0	0	0	4	3	1	0	0	3	3	3
99	Chitra	1	I14019989	1	0	0	0	0	0	0	0	0	0	0	0	0	4	3	0	0	1	3	3	3
100	Kalpana	2	I14020794	1	0	0	0	0	0	1	0	0	0	0	0	1	4	3	1	0	0	4	3	3
101	Priya	1	I14022839	1	0	0	0	0	0	0	0	0	0	0	0	0	3	3	0	0	1	4	3	3
102	Revathi	2	I14023358	1	0	0	0	0	0	0	0	0	0	0	0	0	4	3	1	0	0	3	3	3
103	priyanka	1	I14022526	1	0	0	0	0	0	0	0	0	0	0	0	0	3	3	0	1	0	3	3	3
104	Saranya	1	I14021596	1	0	0	0	0	0	1	0	0	0	0	0	1	3	2	1	0	0	3	2	3
105	Selvi	1	I14022064	1	0	0	0	0	0	0	1	0	0	0	0	1	4	3	1	0	0	3	3	3
106	Sujatha	1	I14022245	1	0	0	0	0	0	0	0	0	0	0	0	0	4	2	1	0	0	2	2	3
107	Uma	2	I14023328	2	0	0	0	0	0	0	0	0	0	0	0	0	3	3	1	0	0	3	3	3
108	Shana	1	I14023477	1	0	0	0	0	0	0	0	0	0	0	0	0	4	3	1	0	0	3	3	3

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109	Divya	1	I14024018	1	0	0	0	0	0	0	0	0	0	0	0	0	4	3	1	0	0	5	3	3
110	Indirani	4	I14024686	1	0	0	0	0	0	1	0	0	0	0	0	1	4	3	0	1	0	4	3	3
111	Shalini	1	I14024757	1	0	0	0	0	1	1	0	0	0	0	0	1	3	3	0	0	1	3	3	3
112	Aruna	2	I14025154	1	0	0	0	0	0	0	0	0	0	0	0	0	3	3	0	1	0	3	3	3
113	lurdhu	1	I14025275	1	0	0	0	0	0	0	0	0	0	1	0	1	3	3	0	0	1	4	3	3
114	sudha	1	I14025706	1	0	0	0	0	0	0	0	0	0	0	1	1	3	3	0	0	1	3	3	3
115	suganya	2	I14025934	2	0	0	0	0	0	0	0	0	0	0	0	0	4	3	1	0	0	3	3	3
116	Kowshika	1	I14027063	1	0	0	0	0	0	0	1	0	0	0	1	1	3	3	1	0	0	2	3	3
117	jayardakthi	1	I14028102	1	0	0	0	0	1	1	0	0	0	0	0	1	2	2	0	1	0	2	3	3
118	Sudha	3	I14030412	2	0	0	0	0	0	0	0	0	0	0	0	0	4	3	1	0	0	4	3	3
119	Gnanapriya	1	I14030454	1	0	0	0	0	0	0	0	0	0	0	0	0	3	3	1	0	0	3	3	3
120	Poonam	3	I14030616	2	0	0	0	0	0	0	1	0	0	0	0	1	4	3	1	0	0	3	3	3
121	Padma	1	I14036144	1	0	0	0	0	0	0	0	0	0	0	0	0	4	3	0	1	0	4	3	3
122	Kiruba	1	I14035009	1	0	0	0	0	0	1	0	0	0	0	0	1	4	3	0	1	0	3	3	3
123	Nithya	2	I14036919	1	0	0	0	0	0	0	0	0	0	0	0	0	3	3	0	0	1	3	3	3
124	Logeshwari	3	I14037060	2	0	0	0	0	0	1	0	0	0	0	0	1	4	3	1	0	0	3	3	3
125	Elizhil	1	I14036774	1	0	0	0	0	0	0	0	0	0	1	0	1	3	3	0	0	1	4	3	3
126	Shobana	3	I15001651	2	0	0	0	0	0	0	0	0	0	0	0	0	4	3	1	0	0	4	3	3
127	Rohini	1	I15002085	1	0	0	0	0	0	0	0	0	0	0	0	0	3	2	1	0	0	2	2	3
128	Mariya	3	I15002199	2	0	0	0	0	0	0	0	0	0	0	0	0	4	3	1	0	0	5	3	3
129	raeshwari	1	I15002858	1	0	0	0	0	0	0	0	0	0	0	1	1	2	3	1	0	0	2	3	3
130	soundarya	2	I15004211	1	0	0	0	0	0	0	0	1	0	0	0	1	4	3	0	1	0	2	3	3
131	geetha	3	I15004705	2	0	0	0	0	0	0	1	0	0	0	0	1	3	3	1	0	0	2	3	3
132	jayashree	1	I15005144	1	0	0	0	0	0	0	0	0	0	1	0	1	3	3	0	0	1	3	3	3
133	tamilselvi	1	I15007258	1	0	0	0	0	0	0	0	0	0	0	0	0	4	3	0	0	1	2	3	3
134	Padma	1	I15007126	1	0	0	0	0	0	0	1	0	0	0	0	1	3	3	0	1	0	3	3	3
135	arthi	3	I15007511	2	0	0	0	0	1	0	0	0	0	0	1	1	3	3	1	0	0	2	3	3
136	shalini	1	I15008170	1	0	0	0	0	0	0	0	0	0	0	0	0	4	2	1	0	0	2	2	3
137	nithya	3	I15008800	2	0	0	0	0	1	1	0	0	0	0	0	1	4	3	1	0	0	4	3	3
138	Nithya	2	I15009464	1	0	0	0	0	1	0	0	0	0	0	0	1	3	3	1	0	0	2	3	3
139	ramya	2	I15009491	1	0	0	0	0	0	0	0	0	0	0	1	1	3	3	0	1	0	2	2	3
140	mohana	1	I15009798	1	0	0	0	0	0	0	0	0	0	0	0	0	4	3	1	0	0	4	3	3
141	nithya	2	I15009997	1	0	0	0	0	0	0	0	0	0	0	0	0	2	3	1	0	0	3	3	3
142	Dhanu	2	I15009486	1	0	0	0	0	0	0	0	0	0	0	0	0	3	3	0	1	0	4	3	3
143	athilakshm	3	I15009839	2	0	0	0	0	0	0	0	0	0	0	0	0	5	3	1	0	0	5	3	3
144	nishana	3	I15009975	2	0	0	0	0	1	0	1	0	0	0	1	1	2	3	1	0	0	2	3	3
145	Vaitheshwa	3	i15010065	2	0	0	0	0	0	0	0	0	0	0	1	1	2	3	1	0	0	2	2	3
146	sudha	2	I15010429	2	0	0	0	0	0	0	0	0	0	0	0	0	4	3	0	1	0	4	3	3
147	geetha	1	I15010947	1	0	0	0	0	0	0	0	0	0	1	0	1	3	3	0	1	0	3	2	3
148	nithya	2	i15011159	1	0	0	0	0	0	0	0	0	0	0	0	0	3	2	1	0	0	2	2	3
149	Rishana	3	I15011414	2	0	0	0	0	0	0	0	0	0	0	0	0	4	3	1	0	0	4	3	3
150	Kalpana	3	I15011945	2	0	0	0	0	0	0	0	0	0	0	1	1	2	3	1	0	0	3	3	3
151	hima	1	I15012134	1	0	0	0	0	0	0	0	0	0	0	0	0	3	3	0	1	0	3	3	3
152	bharathi	4	I15012268	2	0	0	0	0	0	0	0	0	0	0	1	1	2	3	0	0	1	2	3	3
153	rahumathi	4	I15012207	1	0	0	0	0	0	0	0	0	0	0	0	0	3	3	0	1	0	4	3	3
154	uma	1	I15012327	1	0	0	0	0	0	0	0	0	0	0	0	0	3	3	0	0	1	3	3	3
155	indhumath	1	I15012547	1	0	0	0	0	0	0	0	0	0	0	0	0	4	3	1	0	0	3	3	3
156	mallika	3	I15005724	2	0	0	0	0	0	0	0	0	0	0	0	0	4	3	1	0	0	3	3	3
157	saranya	1	I15005915	1	0	0	0	0	0	0	0	0	0	0	0	0	4	3	1	0	0	4	3	3
158	nisha	3	I15006395	2	0	0	0	0	0	0	0	0	0	0	0	0	4	3	1	0	0	3	3	3
159	sangeetha	2	I15006971	1	0	0	0	0	0	0	0	0	0	0	0	0	4	3	1	0	0	3	3	3
160	banumathi	2	I15008867	1	0	0	0	0	0	0	0	0	0	0	0	0	4	3	1	0	0	4	3	3

VARIABLES

AGE

1 – 20-25yrs

2 – 26-30yrs

3 – 31-35yrs

4 – 36-40yrs

SCORE

1 – Primigravida

2 – multigravida

GA OF ADMISSION WITH HG

1 – 0-5WKS

2 – 6-10WKS

3 – 11-15WKS

4 – 16-20WKS

5 – 21-30wks

6 – 31- 40wks

NO OF EPISODES

1 – 1-5 EPISODES

2 – 6 – 10 EPISODES

3 – 11-15 EPISODES

DEHYDRATION

0 – No

1 – yes

OLIGOAMNIOS

0- No

1 - yes

KETONES

0- Neg

1- 1+

2- 2+

3- 3+

ANEMIA

0-No

1 – yes

GDM

0 –No

1 –yes

GGI

0 – No

1 – yes

GEST HT

0 – No

1 – yes

IUGR

0-No

1 - yes

GA AT DELIVERY

1-early preterm

2 –late preterm

3 – term

WGT GAIN

0- no wgt gain

1 -3-5kgs

2 – 6-8kgs

3 – 9-11kgs

4 – 12-14kgs

5 – 15-18kgs

6 – 19-21kgs

BIRTH WGT

1 -1.5-2kgs

2-2.1-2.5kgs

3- 2.6-3kgs

4-3.5-4kgs

NVD

0-No

1 – yes

INSTRUMENTAL

0-no

1 -yes

LSCS

0-No

1 – yes

APGAR 1ST &5TH MIN

1 -0 -3

2 – 4-6

3 – 7-10